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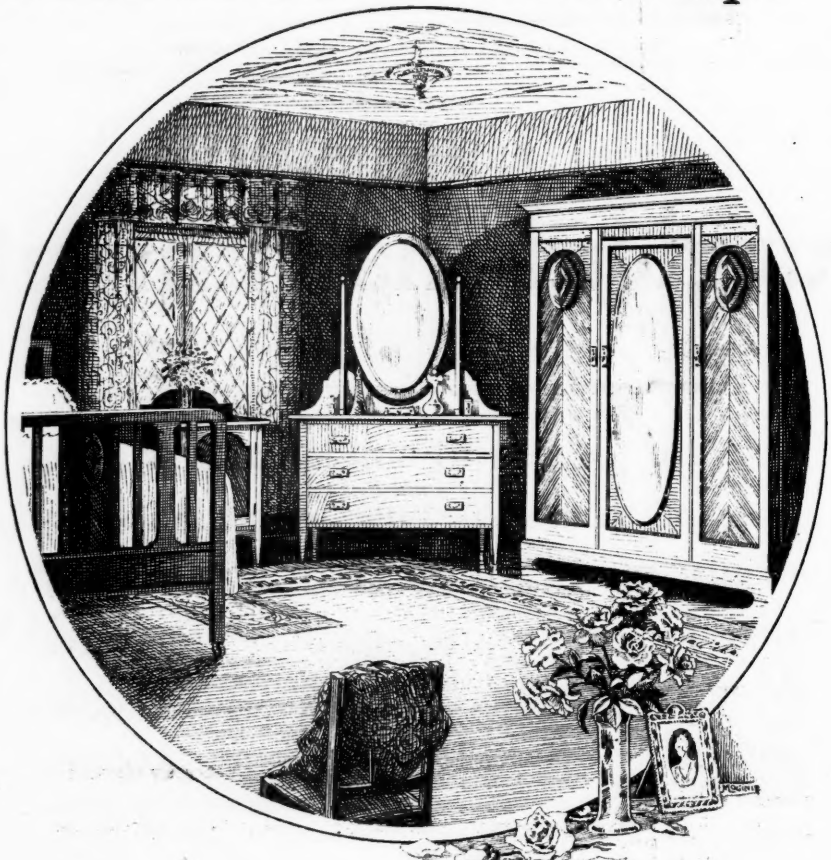

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CONVULSIONS IN CHILDHOOD.[1]

BY A. JEFFERIS TURNER, M.D., M.R.C.S. (ENG.), D.P.H. (CAMB.),

Visiting Medical Officer, Diamantina Hospital for Chronic Diseases, Brisbane.

AN attack of convulsions, an epileptic fit, an eclamptic seizure, all these are caused by a generalized discharge from the motor areas of the cerebral cortex. They are essentially the same thing and you should think of them together, remembering that to give things different names does not make them different. There are, of course, in these seizures infinite varieties in character and degree, which are sometimes suggestive, but very often misleading; you must be very cautious of drawing conclusions either from the severity or localization of the attack. The intensity or distribution of the cerebral discharge may vary greatly in different attacks in the same patient. You must not, for instance, conclude that a convulsive seizure limited to one side of the body is necessarily due to an organic cerebral lesion, for, if you do so, you will often be wrong.

The Cause of the Seizure.

As practitioners in dealing with a case of convulsions, you are concerned with prognosis and treatment. Is this attack dangerous to life or may it be negligible from that point of view? Is it an isolated incident or are the attacks likely to become habitual? Ask yourself, therefore, what are the underlying conditions that determine the attack; are they persistent; are they remediable? Always bear in mind that the causation is two-sided; on the one side is the more or less excitable cerebrum, on the other the immediate stimulus which excites the motor discharge. Now the motor cortex in the child is undoubtedly easily betrayed into convulsive discharges; the younger the child is, the more likely is the convulsion to result from a stimulus of slight intensity or easily remediable and the ultimate prognosis of even repeated attacks may be relatively good. On the other hand, the child is more prone to long continued convulsions or series of convulsions, ending in death, and consequently the immediate prognosis of the attack, even when due to some remediable cause, may be relatively bad. Furthermore, children of the same age differ much in cerebral motor stability. Though any child may go into convulsions on sufficient stimulus, some need but little. Convulsions may run in families.

The immediate stimulus may be almost anything. It may be a mechanical interference with the brain from a fall on the head, a cerebral hæmorrhage or tumour or an inflammation, encephalitis or meningitis. It may be a high pyrexia; some young child-

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 7, 1922.

ren always go into convulsions if the temperature exceeds 39.5° C. (103° F.). In others, pyrexia of over 40.5° C. (105° F.) has no such effect. It may be a toxæmia of any sort or a powerful reflex irritation. I shall reserve my discussion of these for a moment and merely remark that one convulsion smooths the path for another; the reflex grows easier with repetition.

Management of the Attack.

You are frequently called out to deal with convulsions in children and it is well to have a small handbag ready packed for these emergencies. It should contain a small bottle of chloroform, an enema syringe, a very fine catheter capable of passing through the nasal passages of an infant, connected by a tube with a small funnel or syringe, some calomel tablets, chloral and bromide, the former in solution, the latter preferably in tablets, and a small needle or trocar suitable for lumbar puncture. Thermometer, hypodermic syringe and stethoscope you will, of course, have with you. If on arrival the child is still convulsed, you will probably find it in a hot bath or lately removed from the bath. I believe the bath is good treatment in the absence of a medical man; but its usefulness is limited. A hot bath, especially with mustard, draws blood to the extremities; it lowers blood pressure and, as the child is raised out of the bath, it increases heat loss and lowers temperature. My first step in such a case is to introduce a thermometer into the rectum. If there is high pyrexia I would apply cold or tepid water to the body and head while the child is in the bath or as it lies on a mackintosh. Having lowered the rectal temperature several degrees, but not below 39° C. (102° F.), as there is usually a further fall after the sponging or bath, I would proceed at once to administer chloroform. No other treatment is so speedy, safe and certain, provided the chloroform is not pushed too far. Usually the twitchings cease after a few minutes' inhalation. Then I would empty the bowels by a large enema, unless they had been already very freely open, pass a nasal catheter into the stomach and so administer a fairly large dose of chloral, 0.3 gramme (five grains) to a child over one year or 0.6 gramme (ten grains)to a child over two, is not too much. This is better than rectal administration of chloral, for one is never sure whether the rectum will absorb or reject the dose. Having seen the child quietly settled, I would leave a few doses of chloral and bromide in case of any return of restlessness or twitching and perhaps also a dose of calomel.

Treatment comes before diagnosis in this instance, but as soon as the crisis subsides, you must inquire into causes. In the first place, inquire into the commonest causes, pyrexia and surfeit. As the violent muscular contractions may of themselves raise the body temperature, careful inquiry must be made whether the child was hot and feverish before the convulsions began, whether it has had a catarrh or cough or had opportunities of eating excessive or unwholesome food. Frequently undigested substances may be recognized in the evacuation, but do not thereby conclude that you have learnt all the excitant causes. A child in convulsions may pass chunks of raw tomato, but may, for all that, be suffering from incipient pneumonia; so do not fail to examine the chest. A careful inquiry into the child's condition during the past forty-eight hours will usually give you a tentative diagnosis, which will probably be confirmed by its condition the following morning. Its relative cerebral excitability may be guessed from the history of previous attacks or attacks in other members of the family or perhaps by your estimation of the nervous stability of the parents.

Chloroform inhalation, followed by chloral, does not always stop the attack and the prognosis is then grave, though not necessarily hopeless. Morphine hypodermically is sometimes of service; the dose should be small and repeated if necessary. To a child of eighteen months I would give two milligrammes (one thirty-second of a grain), to one of two years two and a half milligrammes (one twenty-fourth of a grain) and repeat the dose if necessary once or possibly twice at half hourly intervals. In an obstinate case I should certainly do a lumbar puncture; but since this proceeding came into use I have seen too few cases in the acute stage to form an opinion as to its value. In these long continued refractory cases you may suspect the presence of a cerebral lesion or (in Brisbane) of lead poisoning; but do not conclude that either of these is necessarily present.

The Significance of Convulsions.

Let us now view our subject from another point. Children are brought to you with a history of fits of some kind. Never treat these cases lightly; they should receive the most careful investigation, for the whole future of the child may depend on the advice you give. Very rarely will you be so fortunate as to witness one of the attacks. Only by very careful questioning of the parents can you form a clear mental picture of what actually occurs during each attack; but this you must always do. You will be able to exclude various kinds of "fits," such as emotional or hysterical seizures, which occur occasionally, but not often in children. Evidence of loss of consciousness should always be sought for; this is conclusive of an epileptiform or eclamptic seizure. You will find, however, that cases occur not very infrequently, as I shall show you, of slight epileptic seizures without loss of consciousness. Obtain full particulars of the child's first fit, of any variations of character in the fits, of their frequency and with the greatest care of any symptoms which may immediately follow or precede them. All this requires much time and patience to elicit; these you must not grudge, for on your management of the case may depend whether the child grows up to be an epileptic.

By epilepsy I mean a condition in which convulsive or minor seizures recur more or less frequently without any apparent cause or from exceedingly trivial causes. The generalized motor discharge or minor phenomena which experience has shown to be of the same order, occur without apparent, or on apparently very insufficient, provocation. As to the mechanism of this condition we know very little, but we know that it may be due to congenital ill-development or birth injury, to hereditary insta-

bility or to acquired instability the result of previous attacks. That is to say, the cerebral factor is overwhelmingly preponderant. But in all convulsive seizures in children the cerebral factor plays some part and there is an infinite series of variations between the congenital epileptic and the normal. No hard-and-fast line can be drawn between convulsive seizures due to toxæmic or reflex causes and true epilepsy. The one grades into the other. Not only so, but attacks of convulsions, however caused, more particularly if frequently repeated, are a cause of epilepsy. Here let me beseech you to free yourselves from the bondage of mere names. If you form your ideas of epilepsy from developed cases in adult life, your ideas will be erroneous and your therapeutics paralysed. Clear-cut distinctions between epileptic seizures and convulsive attacks in early life do not exist. Any such attacks may by repetition cause epilepsy; of few attacks in childhood can it be said that they are hopelessly epileptic.

A little experience will teach you that as a general rule the closer convulsive seizures in children approach the picture of a typical epileptic seizure, the less likely they are to induce permanent epilepsy; and that the slighter and less typical they may seem, the more likely they are to do so, but that even these slighter seizures are sometimes amenable to treatment. Still more important in the prognosis are the symptoms that may accompany the attack. If each attack is preceded by a rise of temperature or preceded or immediately followed by evidences of digestive derangement, the prognosis is relatively good. Why the gorging of unsuitable food should so frequently determine an attack of convulsions in early life is not clearly apparent. I doubt very much whether the irritation is a mere reflex. The painful colic of intussusception in an infant rarely has this effect. I suspect the mechanism is toxæmic, that it is due to some blood condition and depends on quantity more than on kind of unsuitable nutriment. However this may be, the sequence is a common experience. Cases of this kind must be treated by careful dieting and, if the transgressions in diet appear to have been slight, the dieting must be extremely careful and rigorous. Where the cerebral instability appears to be great, it must be restrained by the administration of bromides in full doses, from 0.6 gramme to 2.4 grammes (ten to forty grains), according to age and other conditions, daily for long periods without intermission. Children stand bromides well and though they should be watched for possible overdosage, the drug may usually be continued for six or twelve months or longer without any harm. Cases occur in which bromides are valueless. I have not so far obtained in these cases sufficient success with other drugs to warrant my bringing them under your notice.

I have said nothing as to the terminal convulsions which may occur, for instance, in gastro-enteritis and are of little practical importance; nor of uræmic convulsions, which are very rare in childhood; nor of rickets, so much insisted on by English writers as a predisposing cause, but apparently playing little part here. I will conclude by illustrating my thesis by a few brief abstracts of cases selected from those which I have seen within the last three years. Most of the patients were seen in consultation and I have not been able to follow them up, but some have recently replied to letters of inquiry.

Clinical Reports.

I. Boy, aged two months. His birth had been difficult. He suffered very frequent attacks of rigidity and twitching of the right arm; he had one which I witnessed. He cried during the attack. Occasionally twitching spread to the left arm and sometimes to the legs, in which case general convulsions supervened. The diagnosis was brain injury during birth. The prognosis was bad.

II. Boy, aged one year and ten months. He had curious "turns," several occurring in a day. He stiffened or sat down, became pale, swallowed, sometimes raised his right arm and pronated his hand; his eyes were open and staring. He was quiet after the attack. The patient was the first-born of a family. He was restless, noisy and destructive. He did not talk. The prognosis was bad.

III. Girl, aged ten months. She appeared to be well nourished and healthy in every other way, but had curious "turns." At first they appeared only when she was put into her bath, but later they came when she awakened from sleep. The attacks used to be daily; later they occurred once or twice a week. The patient became stiff, the legs extended, the arms were clenched, the head was thrown back, the mouth and eyes were kept open. This condition lasted for two seconds. The child then gave a cry; afterwards it became limp and inclined to doze. The prognosis was doubtful, but she may improve under bromide.

IV. Boy, aged one year and ten months. He looked healthy and well nourished. He had never had convulsions. The first seizure occurred at the age of twelve months. Later they occurred once or twice a week, but the patient had been free for a month at a time. He cried out "Mamma!"; then tumbled down. There were no twitchings nor stiffness; the eyes were open; he appeared to be dazed or unconscious. He had been very unsuitably fed; he had been given little milk or milk food, bread and butter between meals, etc.. As the attacks came on with artificial feeding and this has been bad, there was good hope of improvement with careful dieting.

V. Minor seizures following severe convulsions at the age of four years. The patient, a girl, was eight years of age. The attacks always occurred at night. She awakened from sleep, making a curious noise. Her eyes were fixed, her lips twitched and turned up, her face turned to the right, her right arm became flexed to a right angle and her right hand twitched. About three seizures occurred each week. They were worse if the patient were excited or went to school. She was ordered to stay away from school and to avoid excitement. She was given plain food, none after 5 p.m.. Bromide of potash (0.5 gramme, *i.e.*, seven and a half grains) was given at 6 p.m.. Two weeks later she had had no attacks. During the next month she had only one attack. I have not heard of this patient since.

VI. Boy, aged two years and three months. Six months age he had a fit, screamed, fell, became rigid, with eyes staring, etc.. Three months later he had another. Altogether, he had five fits. After the attacks he slept for an hour or so. When he awakened he was feverish. "He was always feverish before the attack." He seemed to have been simply fed. He was ordered a strict diet and 0.3 gramme (five grains) of bromide of potash twice daily. Nearly two years later the mother wrote that the boy had had no more attacks.

VII. Girl, aged five years. She had had three seizures with dyspeptic symptoms. There was a bad heredity. She was a bright, nervous child with symptoms of adenoids. Her first attack had taken place six months before. She had become unconscious and stiff; then her muscles jerked and she ground her teeth. She attempted to vomit before each attack. She was noticed to be feverish before the third attack. Her mother had had epileptic fits from the age of eleven until she was twenty-

two. She had given a scream and had gone off into convulsions; she had had none since the birth of her first child. This was the youngest; the older children were adults; some were married with children. She was ordered 0.6 gramme (ten grains) of bromide of soda. Ten months later she had had no further attacks. She was put on a careful diet without medicine. She remained free from attacks for twelve months. She was then badly overfed—five meals a day. The attacks returned and became more severe.

VIII. Girl, aged four years. She was supposed to have had "meningitis" at fourteen months. Since then she had been subject to "turns" after eating anything unwholesome. Sometimes she was feverish; usually she was dull and heavy before the attack. She seemed unconscious, with her mouth clenched, her eyes open and staring. She soon recovered in a hot bath. She had remained free from attacks for two years, but has had two slight "turns" lately.

IX. Minor epilepsy; girl, aged nine years. This patient was apparently healthy and doing well at school. At ten months she had repeated attacks of convulsions for a week and lost the use of her arm for two weeks. She had three or four "turns" in a day. She ran to her mother, saying that she felt sick; her eyes were fixed and staring; she did not answer when spoken to. After a moment she flushed red, became tired and often went to sleep. Her mother's father, aged seventy-one, had had epileptic fits for eight years. Two years later her mother wrote that she still had a slight "turn" occasionally, but on the whole had improved.

X. Boy, aged eleven years. He had minor attacks without loss of consciousness associated with diet. His first attack had occurred two years before, without apparent cause except constipation for three days. The second attack took place a year later. After each attack he was in bed with a "gastric attack," vomiting green slime. Since then he had had many attacks with intervals of a week to three months. He had jerky movements of the head, eyes and arms. The last three attacks had been on the right side only. During an attack he said: "Stop my arm, mother! Don't let it go!" After birth he had appeared paralysed in the right arm and leg for three days and the right arm had always been weaker than the left. He had a big appetite and was excitable and "bad tempered." He was treated by dieting and bromides. No attack had taken place for over a year and then he had a bad one, followed by anorexia and vomiting after eating "Isabella" grapes.

XI. Frequent convulsive seizures always preceded by fever. Girl, aged three years. Her first attack had occurred at twelve months from influenza with high fever. Another had taken place six months later while she was suffering from a heavy cold and feverishness. Since then she had had five attacks, always at night. For instance, she had seemed quite well during the day; at 10 p.m. she was feverish and talking in her sleep; at 3.30 a.m. she cried out and caught hold of her mother. The twitching began in her face and spread to her limbs. Her eyes opened, became fixed and turned upwards. She lost consciousness for about five minutes and vomited before and after the attack. She was strictly dieted and given 0.6 gramme (ten grains) of bromide of potash at bed-time. Six weeks later she had had no further attacks. Eighteen months later her father wrote that she had improved greatly. She had had only one attack, which had occurred three months ago and had ended with vomiting. The strict diet has not been kept up, but they have been careful.

XII. Boy, aged three years, suffered from adenoids. He was backward in intelligence. Six months before, on the first day of a fever, he had had violent convulsions; seven attacks in one night. Two weeks later he had again had fever and convulsions. Since then he had had attacks once a week or in two weeks without fever, some slight, some severe. He had been ordered 0.6 gramme (ten grains) of bromide of soda three times a day. He took this regularly for twelve months and irregularly since. The "fits" immediately disappeared and did not return, but occasionally, at many months' interval, he got slight "turns" in which he was stiff and unconscious for a very short time. The adenoids were removed about two years later. He has grown stronger, but is still mentally deficient.

XIII. Girl, aged six years (recently seen). Twelve months before she had lead poisoning, with colic, severe convulsions and some paralysis of her legs. Other symptoms have cleared up, but she has slight "turns" nearly every week, sometimes several in a day. The attacks always begin in the same way. Sometimes she says: "Mummy, I want to spit up!" Her mouth closes, her lips work, some froth escapes, her eyes are open and staring; she is unconscious. In a very short time her eyes close and she goes to sleep. She awakens in a half of an hour or so fully recovered. I advised bromide (0.6 or 0.9 gramme) twice daily or perhaps potassium boro-tartrate.

XIV. Boy, aged five years. Five months before he had severe convulsions, for which he was given chloroform. A second attack occurred seven weeks later. A few days after the first attack he began to walk badly, dropping his toes. He has slight "lead line" on his gum, opposite his first bicuspid. Apparently all the muscles below the knee, except the calf muscles, are paralysed. There is weakness of the extensors of the fingers, wasting of the thumb muscles. There is a history of powdery paint and nail biting. Gradually the child has made a complete recovery.

XV. Youth, aged eighteen years. He had been subject to fits, which from description appear epileptic, for two years, about once a month. He had been apprenticed to the plumbing trade, but later lived at home and did odd jobs. The attacks always occurred in the morning, usually on Sunday morning. He went to a dance and had a late supper every Saturday night. He was advised to avoid late hours and suppers. He was to take 1.8 grammes (thirty grains) of mixed bromides at bed-time. Five months later he had had only one attack since first seen.

XVI. Boy, aged two years and six months. This patient had "giddy turns." About four months before his mother noticed him falling as if he were giddy. Sometimes he had attacks several times in succession. When he felt the attacks coming, he said "Merry-go-round!" and ran to his mother frightened. He became pale, his eyes were staring as though he were unconscious. He tried to pick himself up, but tumbled again, evidently giddy. Otherwise he appeared to be healthy, but had urticaria and itchy skin and a voracious appetite. The attacks became less frequent since his diet was reduced and there had been none for a month. He was constipated. He was ordered a strict diet, bromide of soda and magnesium sulphate. The attacks became much fewer. Four months later he was successfully operated on for acute intussusception. After that the attacks became more frequent; about once a week. He was ordered 0.5 gramme (seven and a half grains) of bromide of soda and one gramme (fifteen grains) of magnesium sulphate twice a day. Six weeks later he had had only one "giddy turn." Eighteen months later his mother wrote that he had been free from attacks and was well and strong. His diet is carefully watched. The medicine was continued for nine months and is still occasionally given when he gets irritable.

XVII. Girl, aged three years. She has "giddy turns." She is a healthy child. When she learnt to walk she sometimes fell without losing consciousness, but turned very pale and took an hour or two to recover. There were no twitchings. Gradually these "turns" grew less frequent. She has not had any for over a year, but during the last five months she has had three "turns" of a different character. In the last she tumbled down without evident cause and said that the floor was going up and down and she could not walk. She became very pale, but recovered in a few minutes.

XVIII. Girl, aged two years and nine months. She is a large, fat and overfed child. Since she was one year of age she has had "giddy turns" at intervals varying from one week to three months. She says: "I giddy!" or "House falling, mummy!" She stands still. In one attack only did she fall down. Apparently there is no loss of consciousness. The nature of the attacks is doubtful.

XIX. Girl, aged two and a half years. For the last two or three months she has had curious seizures every day or two, usually in the morning when playing. She runs to her mother or stands still, claps her hands, says "Boo-boo!" then folds her hands over her abdomen, stands with eyes fixed and apparently hears and sees nothing. She does not fall. The attack is over in two minutes. She then is tired and sleepy and cross for the rest of the day.

HAY FEVER.[1]

BY W. N. ROBERTSON, C.B.E., M.B., C.M. (EDINBURGH),
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IN April of this year in THE MEDICAL JOURNAL OF AUSTRALIA there appeared an article on the treatment of hay fever by immunization to proteins such as pollen, animal exhalations, etc.. What struck me in regard to this communication was the amount of work involved in testing a patient with various pollens, foods, animal hair, bacteria and so on and the poverty of the results of treatment. Further, many of these patients do not show any sensitivity to these tests.

This led me to think back over the results I had obtained by intranasal treatment of cases that might be classed somewhat loosely as hay fever. May I say here that the term hay fever does not really cover all the cases I propose to bring before you to-night. The term paroxysmal sneezing might be used to describe most of them, although many are apparently of the anaphylactic type, as hay fever or hay asthma.

It is interesting to note that this disease, as far as we know, did not occur in ancient times; indeed, it seems to be a very modern scourge.

Isolated cases were mentioned in lay literature, but Bostock gave the first account on hay fever in 1819.[1] In 1828 he published further observations[2] and McCulloch[3] speaks of a "well known disorder" and states that the term hay fever had lately become fashionable. It would be useless to pursue this history further at this juncture, but an interesting account may be found in a monograph by Morrell Mackenzie (1885), to which I am indebted for these dates.

Mackenzie also states that the English and Americans (and we may now say Australians) are the only sufferers from these complaints. In Norway, Sweden and Denmark it is scarcely ever seen. And it rarely affects the natives of France, Germany, Russia, Italy or Spain. In Asia and Africa it is only the British who suffer. In America it occurs in nearly every State.

In an address delivered before the Ear, Nose and Throat Section of the Australasian Medical Congress, Sydney, 1911, under the caption of "Asthma and Allied Neuroses," I discussed this subject, giving results of intranasal treatment.

For the purposes of this paper I did not endeavour to delve into my notes of the last twenty odd years, but contented myself with reviewing the cases I had seen in the last two years. These amount to one hundred and thirty-five. Unfortunately, in thirty-five the notes are too sketchy to be of any real use, which leaves me to deal with the convenient number of one hundred.

I have grouped these cases under various headings—age, type, local lesion, treatment, result, etc.—and I propose to deal with the cases on these lines, with a few other points, and later to give my views on the directions in which an attack may be made to relieve this troublesome condition.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 7, 1922.

Sex.

My patients comprised forty-five males and fifty-six females.

Family History.

I have recorded a direct family history of hay fever or asthma in 21% of the cases, but have not noted other allied manifestations, as eczema, migraine, angio-neurotic œdema, *laryngismus stridulus*, etc..

Age.

It is notable that young people are the commonest victims. The average age of the sixty-nine patients whose age I have noted is just over twenty-seven. The two youngest were four and a half and five years. Only three patients were over fifty; their ages were fifty-five, sixty-three and sixty-six.

Type.

The predominating type was the classical hay fever, where, annually, chiefly in the spring and early summer, patients came along complaining that they were prostrated with attacks of sneezing, running to twenty, thirty or forty times, when they went into the garden, made a bed, dusted a room, etc..

Another group of patients complained that the moment their feet touched the floor on getting out of bed, they had a severe attack; others again would respond to the slamming of a door or a sudden noise or to a cold draught of air; others, probably the subjects of errors of refraction or astigmatism, reacted to a bright light, say, on stepping out of doors into the sunlight. It is interesting to note in how many of these patients the condition gradually merges into definite asthma. Indeed, the great bulk of asthma patients give a previous history of paroxysmal sneezing.

Local Condition.

In nearly every patient seen during the course of an attack the mucosa of the nose is tumid, pale and watery, whatever other departure from the normal may be present. When examining a nose as a routine measure, not for hay fever, on seeing this typical condition, I have asked: "Do you sneeze much?" and the reply has been "Yes!" "How often?" "About twenty times in succession." Many patients who consult me for defective hearing, exhibit this condition and get relief by treatment of the nose.

The obstruction and catarrh have a very definite, harmful effect upon the ears.

Many persons suffer from deflection and spurs of the septum or from enlargement of the anterior and posterior ends of the inferior and middle turbinals or from the two conditions combined.

Symptoms.

Sneezing, lachrymation, itching of the eyes, palate, pharynx and ears are constant symptoms. The lids

are puffy and congested and, if the case is of the hay asthma type, wheezing, shortness of breath and disturbed rest become manifest. It is interesting that few of the patients who complain of sneezing alone, have disturbed nights. I have seen a few.

Treatment.

On seeing a patient for the first time, I almost invariably attempt to bring about relief of symptoms and tumidity of the mucosa by the use of the cautery, repeated in a week on the opposite side. Then, if the result is not satisfactory, recourse is had to more radical measures if any gross lesion is present, such as a deflected septum, projecting shelf or spur or hypertrophied turbinals, middle or lower.

I may say here that I have a great respect for the turbinal and consider a turbinectomy a crime, except in the last extremity. One can resect a septum and give nasal freedom without damaging any vitally functioning tissue in the nose, but the turbinal is another matter. Even if the soft tissues are reflected and mainly bone is removed, the results are not very useful and often damaging. The moriform posterior ends, of course, are removed. I occasionally snare a middle turbinal if the pressure is great, with satisfactory results, but I do not go out of my way to do it. Polypi I remove and clear any affected sinuses.

It is interesting to note the tremendous reduction in the swelling of the mucous membrane from the use of the cautery. This is far in excess of any actual physical reduction by scarring of the burn. In a few cases the mucous membrane seems to resent the interference and to react the other way for a few days, though the sneezing diminishes.

What does one do with the cautery? One or two applications of the fine wire pointed cautery are made to the septal mucosa from in front of the middle turbinal and extending upwards and backwards as far as one can see, naturally keeping clear of the cribriform plate. These are light touches, no attempt being made to shrink the tissue by burning a groove or rut into it. If the inferior turbinal is much swollen, as it usually is, one groove about the depth of the cautery wire is made along it from back to front, or the fine point is plunged into the substance of the soft tissue and the puncture sealed with iron alum and "Formidin." This controls any bleeding that might result from too hot a point and is an efficient antiseptic plug for the little wound. There is very little reaction by this method and no part of the mucous membrane is damaged.

Surgical interference, apart from cauterization, was undertaken in twenty-five cases. These were mainly resection of the septum, snare to three middle turbinals and treatment of three polypi. In the twenty-two other cases it was determined to apply surgical treatment if the cautery were not successful. As this gave relief, nothing further was done. The relief of sinus disease has a beneficial influence in these conditions.

The number of treatments applied averages just under four. Forty-two patients were made comfortable after two cauterizations, that is, a touch to each side of the septum and probably to the inferior turbinal.

Resection of the septum alone, when the deformity is marked, is rarely effective in securing complete comfort, but in a few cases, where the pressure was great, this operation has been sufficient.

I find the policy of making haste slowly is a good one. Some men cauterize too savagely and too frequently, with consequent severe reaction, sepsis and subsequent scarring. A well-judged application to the alternate sides of the septum weekly is sufficient.

What is the underlying source of this trouble? I have long argued that asthma, hay fever, *angina pectoris* and their allies are due to a disturbance of the vaso-motor mechanism, but I am also sure that this does not take us far enough back, though I am still satisfied that it is one step on the road. Why, for instance, should an asthmatic or hay fever subject show a definite eosinophilia during an attack? Does the anaphylactic theory explain this or have we to go further back still and ask if some defect in the endocrine secretions is the basis? A great deal of work has been done, both in Europe and America, in testing sensitivity to pollens, proteins, etc., and also in the treatment of these conditions with endocrine glands. I have recently read a most interesting article by Grant Selfridge, of San Francisco, on this subject and he makes out a good case against faulty basal metabolism and unbalanced ductless glands.

Janney emphasizes the liability to infection in hypo-thyreoidism. Engelbach and Tierney pillory the hypo-pituitary subject in the same way. Laurence (*Boston Medical and Surgical Journal*, August 5, 1920) refers to a patient with hay fever who later developed asthma, was sensitive to certain foods and who was able to take foods that gave skin reaction on being exhibited whole lobe pituitary substance and who promptly developed the old symptoms when she ceased to take the gland.

I shall not venture further into this subject with which I am not sufficiently familiar, but would but call attention to the fact that we are getting nearer to the first principles of this disorder, hence, nearer to relief for our patients.

This brings me back to the reason why I am troubling you with these notes; I cannot call them a paper. I felt that, though nasal interference does not get quite to the root of the matter, it affords relief to many miserable sinners and I have seen hundreds of them in these twenty-three years. It requires no elaborate laboratory or tedious skin testing, but in a few minutes offers relief to 60% of the sufferers. I believe it has blocked the progression of hay fever into asthma in very many cases and rendered life and work comfortable to very many.

I was interested to find how closely these results tallied with those published in the *Transactions of the Australasian Medical Congress*, Sydney, 1911:

Now there are 60% quite well, 25% much better, 15% no better; then there were 64% quite well, 27% better, 9% no better.

You may say that I have chosen late statistics and the time is too short to be of value, but at some

later time, if I have the leisure, I shall wade back through them all. But I trust that I have said sufficient at this juncture to cause everyone to undertake seriously this simple procedure for hay fever and also to bear in mind the anaphylactic and endocrine ætiology, which is steadily becoming more obvious, thanks to the investigations to which I have alluded.

References.

(1) Bostock: *Transactions of the Medico-Chirurgical Society*, 1819, Volume X., Part I., pages 181 *et sequentes*.
(2) Bostock: *Transactions of the Medico-Chirurgical Society*, 1828, Volume XIV., Part II., page 437.
(3) McCulloch: "Remittent and Intermittent Diseases," 1828, Volume I., page 394.
(4) Grant Selfridge: *Laryngoscope*, February, 1922.

PERITONEAL ECHINOCOCCOSIS.

AN ANALYSIS OF TWENTY-FIVE CASES.

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ECHINOCOCCAL infestation of the peritoneum is a relatively uncommon disease. Of three hundred patients with hydatid disease treated at the Melbourne Hospital during the past thirteen years, twenty-five (8.3%) presented peritoneal involvement.

In this period many of these unfortunate patients were repeatedly subjected to operation. Their case histories show conclusively that multiple infestation of the peritoneum is almost always preceded by rupture of an intra-abdominal hydatid cyst, either spontaneously or by external trauma or during surgical intervention (usually on hepatic cysts). Dévé(1) claims that cysts may develope from scolices implanted in the tissues of the host, either directly in wounds or serous sacs, such as the peritoneum, or more widely *viâ* the blood stream. The clinical observations in this series entirely support the experimental data and emphasize the necessity for technical skill in operative procedure to prevent soiling of the peritoneum with cyst contents. Considering the grave prognosis of multiple echinococcosis of the peritoneum, as much care should be exercised in operations on intra-abdominal hydatid cysts to prevent further soiling of the peritoneum as is universally practised when a localized intraperitoneal abscess is present.

The Mechanism of Peritoneal Infestation.

The mechanism of peritoneal infestation differs with single or multiple cysts.

The various routes of infestation are as follows:

(*a*) The blood stream.
(*b*) Active migration of the embryo.
(*c*) Local implantation secondary to rupture or leakage of a primary intra-abdominal cyst, usually in the liver.

I.—MULTIPLE CYSTS.

(a) By the Blood Stream.

From the stomach the little embryo may pass by the blood stream through liver and lung to the peritoneum. This must be of very rare occurrence with multiple cysts; otherwise it is difficult to explain the comparative infrequency of pulmonary involvement in such cases (11.8% in this series), when many embryos would pass through the lungs to reach the peritoneum.

(b) Active Migration of the Embryo.

The embryo, after piercing the wall of any part of the intestinal tract from stomach to rectum or after piercing the liver if it has reached there, may pass to the peritoneal cavity, whence by active migration or by gravity it may find its way to the lower portion of the abdomen and there develope into a cyst. It seems unlikely that numerous embryos would follow this course in the one patient and multiple cysts probably do not arise in this fashion. There is no evidence of exogenous budding occurring frequently in the peritoneal cavity of man, though, if such a condition did occur, multiple peritoneal cysts might arise from a single embryo.

(c) Local Implantation Secondary to Rupture of a Cyst.

Peritoneal infestation may occur by the escape of fluid containing scolices, brood capsules or daughter cysts from a ruptured abdominal cyst. Numerous scolices or daughter cysts thus become engrafted on the peritoneum, especially over the lower portions of the abdomen, where multiple cysts are so frequently found.

Dévé(1) strongly urges the view that cysts may develope from scolices. He claims to have produced pulmonary embolic cysts after injecting hydatid material into the vein of a rabbit(1) and embolic peripheral cysts following the injection of scolices into the carotid artery.(2) (3) In the old operation scar cysts which must have been implanted there at a previous operation by contamination of the wound surfaces with scolices or other contents of the cyst, are often found. Of the patients with multiple peritoneal infestation 88.2% gave a history of previous initial operation for abdominal hydatid disease—practically always hepatic hydatid—or of some previous acute abdominal crisis often traumatic in origin or of both.

In only two of the seventeen patients with multiple peritoneal cysts were there no previous operation and no note of any earlier abdominal crisis. Probably in both of these cases a history of some previous acute abdominal trouble would have been elicited had it been sought.

The fact that about nine in every ten patients with multiple cysts give a history of previous abdominal operation or crisis suggests strongly that the usual mode of multiple peritoneal infestation is by the sowing of numerous scolices or daughter cysts from a ruptured abdominal hydatid cyst.

II.—SINGLE CYSTS.

(a) By Blood Stream.

In the case of single peritoneal cysts the embryo may be conveyed by the blood stream to the peritoneum.

Under these circumstances it would be expected that cysts would probably be found also in the liver or lung. Of the eight patients with single cysts,

only one showed definite evidence of hepatic infestation at the time of examination, but two others had been operated on some years previously for liver hydatids. No pulmonary cysts were found in any of these patients. It would therefore seem probable that conveyance by the blood stream was only an occasional mode of infestation of the peritoneum with single hydatid cysts.

(b) Active Migration of the Embryo.

This would appear to be the usual method by which the little embryo reaches the peritoneal cavity. The wall of any part of the alimentary tract from stomach to rectum or the liver itself may be pierced by the parasite, which, on reaching the peritoneum, developes into a single cyst. Only in this way is it possible to account for the absence of associated pulmonary involvement and the infrequency of hepatic infestation in these cases.

(c) Local Implantation Following Rupture of a Cyst.

None of the patients with single cysts gave a history of any previous acute abdominal trouble. Two had been operated on for hepatic hydatids fifteen and five years before respectively. In these cases the peritoneal cyst was probably part of the original hydatid infestation and had no relation to the previous operation. In the former the cyst was greatly thickened and almost cartilaginous, suggesting that the parasite had been present for some considerable time; in the latter a huge, suppurating cyst extended from the under surface of the liver to the right iliac fossa in such a position as to suggest that it had originally been an hepatic cyst which was extruded into the peritoneal cavity in the course of its development. Such an occurrence might occasionally lead to the production of a single peritoneal hydatid cyst.

It is difficult to conceive how only one cyst would develope if the peritoneal cavity had been soiled by fluid containing numerous scolices and daughter cysts—conditions which must almost invariably lead to multiple infestation.

In the case of single peritoneal cysts the parasite as a rule probably reaches the peritoneum by active migration through the wall of some part of the gastro-intestinal tract or the liver if it has previously reached there, while occasionally the embryo may pass to its destination *viâ* the blood stream.

The Pathological Condition of the Cysts.

The pathological condition of the cysts is shown in Protocol No. 1.

It will be seen that retrogressive changes were present in the cysts in 20% of these patients.

Symptomatology.

The various clinical features presented by this series of cases may now be considered in some detail.

The age and sex incidence of the patients are shown in the accompanying protocol (No. 2) and need little comment.

PROTOCOL No. 2.—AGE AND SEX INCIDENCE.

Age.	Male.	Female.	Total.
15-20	—	—	—
21-30	6	3	9
31-40	3	3	6
41-50	1	3	4
51-60	3	1	4
61-80	2	—	2
Total ..	15	10	25

The clinical picture of peritoneal echinococcosis differs not only according as the infestation is single or multiple, but also according to the presence or absence of complications such as suppuration or rupture.

I.—UNCOMPLICATED MULTIPLE PERITONEAL CYSTS.

Past History.

In the majority of cases (88.2% in this series) there is a history of previous operation for abdominal hydatids, usually hepatic, or of some acute abdominal crisis following rupture of a cyst into the peritoneal cavity.

The interval until the patient with signs of multiple peritoneal infestation again seeks medical advice usually varies from a few months to five years, though in one instance where multiple calcified cysts were found the previous trouble dated back thirty-two years.

Onset.

The onset of symptoms is usually slow and insidious; only rarely does the acute onset of complications (rupture or suppuration), causing systemic manifestations, lead to the discovery of the disease.

Symptoms.

As a rule, the sole complaint of the patient is that the abdomen has been gradually increasing in

PROTOCOL No. 1.

Cyst.	Number of Cases.	Uncomplicated.	With Complications.			
			Suppuration.	Rupture With Suppuration.	Rupture Without Suppuration.	Retrogression, including Calcification.
Single	8	5	0	1	1	1
Multiple	17	11	2	0	0	4

size or that one or more lumps in the abdomen have been progressively enlarging during the previous few months (generally from three months to three years).

Gastro-intestinal disturbances are uncommon, though anorexia, vomiting, a feeling of fullness in the abdomen or definite pain after meals, colicky pains and constipation are occasional symptoms.

When multiple cysts are present in the pelvis, pressure symptoms may be marked. Frequency of micturition and pain during or after the act may be present, while constipation may be troublesome.

Systemic manifestations are not found in uncomplicated cysts; the temperature, pulse and respiration are unaffected, while weakness and wasting are rarely observed.

The presence of hydatid disease elsewhere in the body may cause the usual symptoms of the disease in such situations to be added to the clinical picture.

On examination the absence of any systemic manifestations is obvious. Locally palpation reveals multiple smooth, firm, rounded, elastic tumours freely movable under the examining hands or even with change of posture of the patient and having no apparent connexion with any of the abdominal viscera. In some cases these masses are easily seen on inspection of the abdomen.

On percussion, the tumours are absolutely dull unless covered by bowel, while a typical hydatid thrill is found more frequently with peritoneal cysts than with cysts elsewhere.

Case No. 11 illustrates the history of these patients with multiple peritoneal infestation:

A male, aged twenty-eight, was admitted on March 14, 1909, with a penetrating bullet wound of the abdomen. At operation an hydatid cyst of the liver was found to have been ruptured by the bullet and the contents of the cyst were spread throughout the peritoneal cavity.

Over four years later, on June 27, 1913, the patient returned, complaining that for the past two years he had suffered from occasional bilious attacks in which he felt dizzy, weak and nauseated, while in the preceding six months several lumps had appeared in the abdomen and there had been occasional attacks of colicky pain. The tumours had the characteristics of peritoneal hydatid cysts and at two operations several cysts were removed.

Within the next eighteen months, two further operations were performed, though some cysts even then had to be left untouched.

A year later, on February 17, 1916, the patient returned complaining that a week previously his palms and soles became very itchy, the irritation gradually spreading all over the body, while the hands and tongue became very swollen. For an hour there was difficulty in breathing. There had been a slight rigor and vomiting had occurred once at the onset.

Fever was present. Two days later a feeling of soreness, which had persisted, was noticed in the abdomen. On examination nothing abnormal could be detected. This was an attack of generalized urticaria almost certainly due to rupture of an hydatid cyst into the peritoneal cavity.

Since this time, the patient has not been examined and efforts to trace him have failed.

Difficulty in the diagnosis of multiple peritoneal cysts is most marked in those rare cases where the onset of the condition is apparently acute and a history of previous abdominal crisis or operation for hydatid is lacking.

II.—Uncomplicated Single Cysts.

As the majority of single peritoneal hydatid cysts are found in the pelvis, the signs and symptoms produced by them are usually those of a simple tumour in the pelvis and are due to pressure of the cyst on the surrounding structures.

(a) In the Male.

In the male, the cyst is most frequently found in the pouch between the bladder and the rectum, hence urinary symptoms—frequency, pain, diminution in power of the stream, retention or overflow incontinence—result from pressure on the bladder, while constipation follows from pressure on the rectum. Examination *per rectum* reveals a tumour, usually fairly large and perhaps semi-fluctuant, in the region of the prostate. The rectum may even be definitely narrowed by the pressure of the cyst.

Enlargement of the prostate is the condition most frequently diagnosed in mistake for a single peritoneal hydatid cyst. This difficulty in diagnosis is illustrated by the history of Case No. 31:

A male, aged seventy-one, complained that for the past eight weeks the urinary stream had been getting much smaller and though previously he had to micturate twice or thrice each night, during this period it was necessary to pass water five times during the night. For three weeks pain after attempts at micturition and overflow incontinence had been present, while the urine had been offensive. The bowels were constipated. On catheterization over a litre of urine was withdrawn. Rectal examination revealed in the region of the prostate a large, rounded, non-tender swelling bulging into the rectum and constricting the lumen of the bowel. Operation revealed a large tumour reaching almost to the anterior wall of the bladder and nearly filling the lower portion of this viscus. On dividing what was thought to be the capsule of the prostate, much fluid escaped and the wall of a large hydatid cyst presented. After evacuation this was closed and the bladder drained. Four days later the patient became drowsy and gradually other manifestations of uræmia supervened. Death occurred on the sixth day after operation. *Post mortem* examination was not permitted.

(b) In the Female.

In the female symptoms in these cases are also due to pressure. The cyst is usually found in the pouch of Douglas and so is easily palpable *per vaginam* or *per rectum*.

Constipation is usually present from pressure on the rectum. Menstrual disorders, though they occur, are uncommon. Occasionally frequency of micturition is present.

In the female a uterine myoma or an ovarian cyst is most commonly confused with a single hydatid cyst. These difficulties in diagnosis are illustrated by the following case histories:

Case No. 35.—Female, aged twenty-seven, was quite well until the last confinement, a year ago, since when she had been weak and listless and had lost weight. Periods had been regular, but the flow was scanty. Pelvic examination showed a hard, slightly tender mass in the posterior fornix, apparently involving the cervix. The diagnosis was probable uterine myoma. At operation a calcified hydatid cyst was removed from the pouch of Douglas.

Case No. 253.—Female, aged fifty-six, gave a good previous history. Two days before admission there was a slight rigor, soon followed by a generalized irritable rash. Vomiting occurred and persisted till operation. Epigastric pain appeared soon after the vomiting, which temporarily eased the pain. The bowels were constipated. Micturition was less frequent after the onset of the pain.

The temperature was 37.5° C. (99.6° F.), the pulse-rate 132, regular, but feeble. A large, globular, movable tumour, dull on percussion, was palpable in the lower abdomen.

A papular rash, irregular in shape and size, was present all over the body.

At operation the hydatid cyst was removed.

Here some fluid must have escaped from the cyst, giving rise to an acute abdominal crisis and a generalized urticaria. The pre-operative diagnosis was torsion of the pedicle of an ovarian cyst. Apparently peritoneal hydatid disease was not considered or more stress would have been laid on the presence of the rash.

III.—Systemic Manifestations in Peritoneal Hydatid Cysts.

It has already been emphasized that the clinical picture produced by uncomplicated cysts is essentially due to the mechanical pressure exerted on adjacent structures and is never characterized by systemic manifestations. When such features occur, they are due either to: (*i.*) Secondary infection, with bacteria producing local sepsis, and toxæmia, or (*ii.*) rupture of the cyst, leading to (*a*) signs of an acute abdominal crisis or (*b*) anaphylaxis due to the absorption by the peritoneum of a toxigenic dose of hydatid fluid in a patient already sensitized.

Though suppuration and rupture of a peritoneal hydatid cyst both produce systemic manifestations, the most common complication of these cysts—retrogressive change—leads to no characteristic alteration in the general or local condition of the patient whereby it may be recognized. If the process goes on to calcification, the increased hardness and resistance of the tumour may suggest the supervention of this complication.

(1) Suppuration of Peritoneal Hydatid Cysts.

Suppuration of a cyst produces the usual local and constitutional symptoms of sepsis—pain and tenderness locally in the tumour, with rigors, sweats, fever, rapid pulse, wasting, weakness and diarrhœa if the condition remains unrelieved. These symptoms are due to the absorption of toxic products from the suppurating cyst and are not found where the cyst wall is so altered as to prevent interchange between the cyst contents and the body fluid.

Rupture of a suppurating cyst leads to peritonitis—usually general—and death frequently follows. The history of a patient with a suppurating peritoneal hydatid cyst is very similar to that of a patient with an intraperitoneal abscess due to any other cause.

No data were obtainable regarding the infecting organism in these cases of suppurating hydatid cysts. The organism may have come *viâ* the blood stream, but probably infection from the adjacent bowel is more common.

(2) Rupture of Peritoneal Hydatid.

Rupture of a peritoneal hydatid cyst may lead to one or other of two typical syndromes:

(*a*) Where there has been a tumour in the abdomen, the disappearance of this tumour with the symptoms of an acute abdominal crisis and the production of urticaria is pathognomonic of rupture of an hydatid cyst. Two such cases have already been cited—No. 11 and No. 253. In the latter case the cyst was leaking so slowly that the tumour was still palpable.

In case No. 290, diagnosed as acute appendicitis, there had apparently been no urticaria, though at operation a ruptured cyst with thick, almost cartilaginous walls was found. The probable explanation of the absence of urticaria lies in the fact that different hydatid fluids possess in varying degree the property of being able to produce urticaria, while patients possess different degrees of sensitiveness.

(*b*) The anaphylactic syndrome is characterized by the sudden onset of signs of an acute abdominal crisis combined with severe collapse. Dyspnœa and cyanosis, vomiting and diarrhœa are usually marked, while the pulse is very rapid and barely perceptible. Tenderness and rigidity of the abdomen may or may not be present. Later an urticarial rash may appear. Death may follow quickly, but more frequently improvement rapidly occurs within a few hours.

Case No. 171 illustrates the dramatic results which may follow rupture of a cyst into the peritoneal cavity:

> Male, aged twenty-one years, was admitted with the history that ten months ago he collapsed suddenly at work and operation revealed a ruptured hydatid cyst of the liver. He was readmitted wihh a similar history of sudden collapse while at work. On examination the patient was found to be very collapsed, with an impalpable pulse at the wrist, marked cyanosis and great thirst. He was unable to answer any questions. After the injection of one cubic centimetre of pituitrin the systolic blood pressure was found to be 108 millimetres. The liver dulness extended four fingers' breadth below the costal margin, while a sinus was present in the old operation scar. The flanks were clear and there was no rigidity or tenderness of the abdomen.
>
> Some hours later the general condition of the patient was much improved, the pulse being stronger and the cyanosis less marked. The systolic blood pressure was 120 millimetres. A slight urticarial rash was present on the abdomen.
>
> A week later there was some pain with tenderness on deep pressure in the epigastrium and the sinus was found to have closed. X-ray examination failed to show anything abnormal about the upper margin of the liver and diaphragm.
>
> A few days later the patient was discharged.

Following rupture of a cyst into the peritoneal cavity, the secondary sowing of multiple cysts usually causes further symptoms in a few months or years.

Should the cyst rupture into surrounding hollow viscera, such as bladder or bowel, or into the vagina, the cyst contents will be passed *per urethram, per rectum* or *per vaginam.*

The Diagnosis of Peritoneal Hydatid Cysts.

Protocol No. 3 shows the pre-operative diagnosis of these twenty-five cases of peritoneal hydatid disease and emphasizes the difficulty of diagnosis in the case of single cysts where there is seldom any history of previous acute abdominal crisis or operation for hydatid disease. In the majority of cases of multiple hydatid infestation of the peritoneum, such a history helps greatly in arriving at the true diagnosis. Thus in fourteen patients with such a history the diagnosis was right in every case, while in only one of three patients without this history was the correct diagnosis made before operation.

PROTOCOL No. 3.—PRE-OPERATIVE DIAGNOSIS OF PERITONEAL HYDATID.

Cyst.	Total Number.	Correct Diagnosis.	Incorrect Diagnosis.		
			Uterus and Adnexa.	Prostate.	Acute Abdominal Crisis
Multiple ..	17	15	2	0	0
Single ..	8[1]	0	2	3	2

It will be seen that in single cysts the presence of complications led in two cases to the diagnosis of acute abdominal crisis, while in the male uncomplicated cysts were mistaken in every case for enlargement of the prostate.

Omental sarcomata and simple mesenteric, omental and pancreatic cysts would very rarely occasion any difficulty in diagnosis.

When the previous history is suggestive or there are signs of echinococcal infestation elsewhere, *e.g.*, liver or lung, the diagnosis is easy.

The character of the tumour is the most helpful single physical sign, being typically a round, tense, freely movable tumour, dull on percussion and occasionally giving the true pathognomonic hydatid thrill.[5]

The presence of a definite eosinophilia is suggestive of this disease.

If the former operation for hydatid took place more than eighteen months previously (thus allowing sufficient time for residual antibody to have disappeared from the blood) a positive hydatid complement fixation test in the blood is practically diagnostic of active hydatid disease somewhere in the body.

X-ray examination, especially if artificial pneumoperitoneum be produced, aids in the localization of any abdominal cyst. Care must be taken to avoid puncturing a cyst when introducing gas into the peritoneal cavity.

Exploratory puncture of a probable hydatid cyst is never justifiable until the hydatid complement fixation test in the blood has failed to yield the reaction. No response to a Casoni intradermal test may be regarded as an indication that the patient is not sensitive to hydatid fluid and therefore, even if an hydatid cyst is present, will not suffer from anaphylactic symptoms after puncture of the cyst. But a reaction to this test gives no assurance that the subsequent leakage of the cyst contents will not cause a secondary sowing of multiple cysts in the peritoneal cavity.

Prognosis.

In the prognosis of a case of peritoneal echinococcosis the result of operative intervention must be considered from the standpoint of the immediate risk involved and of the prospect of ultimate cure.

The immediate prognosis in this series was found to depend on the age of the patient, the situation of the cyst, the presence or absence of complications and the type of operation performed.

The remote prognosis regarding ultimate cure depends on the presence of multiple infestation as well as on the incidence of such complications as rupture prior to or during operation.

[1] In one of these cases a latent peritoneal cyst was found during laparotomy for acute appendicitis.

The immediate prognosis of cases of uncomplicated peritoneal hydatids is good. Death resulted in only one of the seventeen patients with multiple cysts, being due in this case to general peritonitis secondary to a vesical fistula accidentally established at operation during the separation of a calcified cyst from the bladder. Three of the eight patients with single cysts died, one from uræmia, another from general peritonitis secondary to post-operative intestinal obstruction and the third from general peritonitis secondary to rupture of a suppurating cyst. All these patients were over forty-five years old; so youth suggests a more favourable outlook.

The ultimate prognosis regarding cure is bad in patients with multiple cysts, as most of them require several operations for recurrences of the condition. In this series two patients had been operated on once, seven twice and two thrice; two others had undergone five operations, while four had submitted to four, six, eight and eleven laparotomies respectively.

In 75% of the patients with multiple infestation, it was found impossible at operation to remove all cysts. Of these patients 63.8% have been re-operated on in this hospital, while the others have probably either been operated on elsewhere or will return later.

In thirteen (76.5%) of the patients with multiple and in one (12.5%) of those with single peritoneal cysts, parasites were found also in other situations—liver, six; retroperitoneal tissues, three; subcutaneous tissues and lungs, two each; spleen, one.

It is obvious that the prospect of complete cure of patients with multiple peritoneal cysts is remote until a drug lethal to the parasite and harmless to the human host is found. Surgical interference often leads only to many scars, adhesions, incisional herniæ and eventually to death, unless the patient is carried off by some intercurrent disease.

Single peritoneal cysts, on the other hand, are usually comparatively easy to remove completely and here the prognosis is good, provided further cysts are not present in other situations. Of the eight patients in this series, two had previously undergone operation for hydatid of the liver some years before, while in another hepatic infestation was obvious at the time of operation on the peritoneal cyst.

Single cysts (more so than multiple) often remain unnoticed by the patient until the onset of some complication causes medical attention to be obtained.

The prognosis is necessarily less favourable where rupture or suppuration of a cyst has supervened, while the outlook is particularly grave when rupture of a suppurating cyst occurs.

Multiple peritoneal infestation may follow rupture of or operation on a single cyst if the contents of the cyst escape into the peritoneal cavity.

Speaking generally the prognosis of multiple peritoneal cysts is unfavourable, of uncomplicated single cysts is good.

Treatment.

Prophylactic treatment consists in the exercise of the greatest care at operation on an hydatid cyst to prevent contamination of the peritoneum with cyst contents, while exploratory puncture of a probable hydatid cyst in the abdomen should never be attempted until the hydatid complement fixation test in the blood has failed to yield a reaction.

MULTIPLE CYSTS.

The operative treatment of multiple peritoneal cysts has already been shown to be highly unsatisfactory. Six different types of operation were performed on various cysts in these seventeen patients with multiple peritoneal infestation. As in a particular case more than one method was frequently adopted to deal with different cysts, comparison of the various procedures is difficult.

The ideal treatment where possible is complete removal of the cyst, including the adventitia. If it is impossible to separate the cyst entirely from surrounding structures, the adherent portion of the adventitia may be left behind.

As regards immediate mortality, all methods used were satisfactory. The only death in the series with multiple cysts resulted from general peritonitis secondary to tearing of the wall of the bladder while separating it from an adherent cyst.

Protocol No. 4 shows the situation of the cysts and the type of operation to which they were subjected.

SINGLE CYSTS.

Of those with single cysts, not one of the five patients who survived, has returned for further treatment. Complete removal again gave the best results.

Two of the three patients in whom drainage was necessary, died from general peritonitis. In one this condition was present before operation, in the other it was secondary to post-operative intestinal obstruction.

In cases simulating prostatic enlargement, where there has been retention of urine and the renal efficiency is poor, the operation should be done in two stages, first draining the bladder suprapubically and later removing the cyst. In case No. 31, with a single cyst simulating prostatic adenoma, death from uræmia occurred on the sixth day after operation at which the bladder was drained and the cyst removed at the same time.

Complications Following Operation.

The complications following operation may be enumerated as follows:

(*i.*) A reactionary elevation of temperature up to 37.8° C. (100° F.) may be present in the first forty-eight hours.

(*ii.*) A more prolonged pyrexia, usually beginning towards the end of the first week after operation, is generally associated with post-operative infection of the cyst cavity.

(*iii.*) General peritonitis (two cases).

(*iv.*) Intestinal obstruction (two cases).

(*v.*) Persistent sinus (one case).

(*vi.*) Incisional hernia (29.4% of cases), the high incidence probably being due to the great number of incisions and the marked increased in intra-abdominal pressure.

The average duration in hospital after operation of patients with multiple cysts was 30.3 days, with single cysts 23 days (deaths excluded).

An analysis of these case histories suggests that the operation of choice in all cases of peritoneal hydatids is complete removal of the cyst; if this is not possible, the portion of the adventitia adherent to surrounding structures may be left behind. If neither of these procedures be practicable, closure of the cyst after emptying it of its contents and injecting 10% formalin in glycerine into the cavity is advisable.[6]

Only when it is absolutely indicated should drainage be established.

Conclusions.

1. Hydatid disease of the peritoneum is frequently a multiple infestation, seventeen out of twenty-five cases in this series presenting multiple cysts.

2. Retrogressive change—often going on to calcification—is the most frequent and least serious com-

PROTOCOL NO. 4.—THE VARIOUS TYPES OF OPERATION ON PATIENTS WITH MULTIPLE PERITONEAL CYSTS.

Type of Operation.	Omentum.	Pelvis.	Peritoneum.	Total.
Removal—				
(*a*) Complete	13 } 14	6 } 8	5 } 6	24 } 28
(*b*) Portion of the adventitia left	1 }	2 }	1 }	4 }
Closure—				
(*a*) Closure only	—	2 } 2	2 } 3	4 } 5
(*b*) After injection of 10% formalin in glycerine[6]	—	— }	1 }	1 }
Drainage—				
(*a*) Drainage only	— } 1	4 } 4	1 } 4	5 } 9
(*b*) With marsupialization	1 }	— }	3 }	4 }
Total	15	14	13	42

plication of peritoneal hydatid cysts (20% in this series).

3. The clinical data in this paper entirely support the view that local implantation of hydatid elements derived from a leaking or ruptured intra-abdominal cyst, or from the dissemination of cyst contents during operative interference is the general mode of origin of multiple peritoneal echinococcosis. It was actually possible in 88.2% of the cases to prove the existence of a primary operation for intra-abdominal (usually hepatic) hydatid or to elicit the history of an acute abdominal crisis frequently following trauma which may or may not have necessitated operative intervention.

4. Single cysts, on the other hand, probably originate by the active migration of a single embryo from the alimentary tract direct to the peritoneum or perhaps as a metastasis *viâ* the blood stream.

5. The clinical picture of uncomplicated peritoneal hydatid is due to pressure exerted on adjacent structures and is generally characterized by the appearance of palpable cystic tumours. Single cysts are usually located in the pelvis and early in their evolution produce pressure symptoms.

6. Systemic manifestations occur only when some complication supervenes. Rupture or leakage of a cyst may lead to the anaphylactic syndrome, while secondary bacterial infection usually produces the clinical picture of localized intraperitoneal sepsis. Retrogressive changes are, as a rule, unassociated with features of clinical import.

7. The pre-operative diagnosis of multiple peritoneal echinococcosis has been satisfactory in this series, 88.2% of cases being correctly diagnosed. A history of preceding intra-abdominal operation for hydatid should always suggest this possibility. On the other hand, a single cyst is difficult to diagnose. In the male prostatic adenoma and in the female ovarian cyst or uterine myoma are usually suspected. The more universal application of the Casoni intradermal test and the complement fixation reaction in abdominal tumours would enable a correct diagnosis to be made with greater frequency.

8. The immediate prognosis regarding life in operative intervention is dependent on the age of the patient, the accessibility and number of the cysts and the presence of complications such as sepsis or rupture. The prospect of complete cure in single cysts is good, but with multiple infestation cure can rarely be attained, even after many operations.

9. At operation the greatest possible care should be taken to prevent soiling of the peritoneum with cyst contents.

10. In peritoneal hydatid, complete removal of the cyst (including the adventitia) is the surgical ideal; where this is not possible, the adherent adventitia may have to be left *in situ*. Closure of the adventitia after removal of the cyst and its contents and the injection of 10% formalin in glycerine into the cavity yields satisfactory results. Drainage should be employed only where it is definitely indicated.

References.

(1) Dévé, F.: *"De l'échinococcose secondaire embolique," Comptes Rendus de la Société de Biologie*, 1901, June 8, Volume 53, No. 21, page 608.

(2) Dévé, F.: *"Echinococcose secondaire embolique périphérique," Comptes Rendus de la Société de Biologie*, 1913, July 19, Volume 75, No. 27, page 100.

(3) Dévé, F.: *"Echinococcose cérébrale métastatique expérimentale," Comptes Rendus de la Société de Biologie*, 1920, April 24, Volume 83, No. 14, page 511.

(4) Dévé, F.: "Secondary Echinococcus," French Supplement to *The Lancet*, 1919, November 8, Volume II., page 835.

(5) Barnett, L. E.: "Hydatid Thrill," *New Zealand Medical Journal*, 1921, October, Volume 20, page 285.

(6) Kilvington, B.: "An Address," THE MEDICAL JOURNAL OF AUSTRALIA, 1921, December 17, Volume II., Eighth Year, No. 25, page 555.

Reports of Cases.

A CASE OF RING SARCOMA OF THE CILIARY BODY.[1]

BY J. LOCKHART GIBSON, M.D. (EDIN.), M.R.C.S. (ENG.), *Brisbane.*

J.S.H., aged forty-five years, was referred to me by Dr. Douglas on July 29, 1921.

History.

He had noted something wrong with his left eye for a month or five weeks.

Present Condition.

The vision of the right eye was $^6/_6$, while that of the left eye was $^6/_{18}$.

I found a red granulation-like growth in the lower outer quadrant of the anterior chamber, squeezed in between the base of the iris and the periphery of the cornea. A similar growth was found in the inner part of the anterior chamber. Each of these growths presented a convex side towards the pupil and a broad base at the root of the iris. They measured three millimetres at their bases and projected 1.5 millimetres towards the pupil. A very small pin's head growth could be seen at the most inferior part of the anterior chamber upon looking carefully. The tension of the eye was + ½ as compared with the tension of the other eye. A trace of iritic deposit was found on the anterior surface of the lens. The fundus could be clearly seen and showed no disease.

Homatropine enlarged the pupil upwards and downwards, but imperfectly laterally. A grey swelling could then be seen between the base of the iris and the lens in the situation of each of the larger growths. There seemed to be little doubt that I was dealing with a sarcoma of the iris or ciliary body. To exclude a gummatous condition a Wassermann test was done that day by Dr. Duhig, but no reaction was obtained. In this case this result was accepted as evidence excluding gumma. The patient was sensible enough to allow me to remove the eye next day, in spite of the fact that its vision was $^6/_{18}$. The eye was exhibited here at a previous meeting to show the growths in the anterior chamber. It was hardened in 2% formalin and on account of its interest and my desire to have it well mounted, I sent it to Professor Welsh, of Sydney University, for his opinion and in order that it might be mounted in his laboratory. I am able to tell you his opinion and also to show the details of a beautifully mounted specimen.

The growth turned out to be macroscopically colourless, so much so that Professor Welsh made an exhaustive examination to make sure whether it was a glioma and therefore a retinal growth or a sarcoma and therefore growing from mesoblastic tissue. He also kindly asked Dr. Oliver

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 7, 1922.

Latham to stain some sections with this end in view. His report reads as follows:

"No evidence of glia cells was found, nor were the blood vessels of the growth like those commonly found in glioma. The evidence points to sarcoma of connective tissue origin and to an origin from the ciliary body. The cells of the growth are small and mostly of the spindle type, but that is quite consistent with its being a sarcoma. The only difference between it and Figure No. 77 in Pyle's 'System of Ophthalmic Practice' is that Pyle's case was heavily pigmented and yours is very lightly pigmented. The blood vessels of the growth are typical of the more malignant (*i.e.*, more highly cellular) forms of sarcoma, being little more than dilated endothelial tubes supported only by the small cells of the growth itself. An interesting feature is that the tissues of the ciliary body are reacting to the advance of the invading sarcoma cells. This is indicated by the emigration of eosinophile leucocytes in unusual numbers and also by lymphocytes over a zone covering the anterior part of the tumour mass."

I am able to show you to-night that the growth is colourless macroscopically, that it is situated in the ciliary body and that it invaded the anterior chamber through the root of the iris.

You will also see that it is what Pyle calls a ring or annular sarcoma of the ciliary body, as, by carefully examining the specimen you will notice that it occupies the ciliary region of fully the lower half of the eye. I was able to tell Professor Welsh before he completed his report that the patient died, evidently from mestastasis in the brain, within four months of the date on which his eye was enucleated. The chief symptoms during the last two months of his life were headache and vomiting and some falls. He was unconscious and unable to speak for the last week and died on November 12, 1921. Up to a month before and probably up to the time of his death there was no sign of papillœdema in his right eye. There was no return in the orbit of the left eye. Permission to perform a *post mortem* examination was not obtained.

Professor Welsh adds, after hearing of the death of the patient:

"Assuming, as I think we must assume, that the death was due to metastatic growth in the brain, we could not have reached a satisfactory explanation of that development unless we had proof that the primary growth was one possessing a high-grade malignancy. But if, as I agree, it is a melanotic sarcoma, that fact would explain why the patient could have been the subject of a secondary growth while the primary growth was still so small."

Pye, quoting J. B. Lawford and Treacher Collins,[1] writes: "Patients much more often die of metastatic growths than of recurrence in the orbit. They are most often found in the viscera, chiefly in the liver and lungs. In the majority of cases a patient with metastasis succumbs to their effects within three years from the date of excision of the eye. Much longer intervals may, however, elapse."

It will be seen, therefore, how highly malignant the growth in my patient was and how important it was to remove the eye at once and give him every chance of life. Fortunately I did this.

Reviews.

SEXUAL PROBLEMS.

THE second edition of Mr. K. Menzies's book on masturbation, which treats of the subject from the psycho-analyst's point of view, is practically a reprint of the first edition.[2]

The disciples of Freud attach such importance to the sex impulse and regard it in so broad a sense that they see its influence in most, if not all, forms of human conduct. The contents of Mr. Menzies's book, "Auto-Erotic Phenomena in Adolescence," seem to show that he is no exception to the rule. He sees in the masturbator the result of a desire to continue enjoyment found in infancy to accompany stimulation of various erogenous zones. At puberty these pleasure grounds are forsaken for the greatly enhanced pleasure to be obtained by manipulation of the genitalia. He sheets home the blame for the phenomenon of masturbator's remorse to mental conflicts with such fanciful origins as fear of being deprived of the favourite and principal joy-bringing plaything (castration complex) and unconscious association between indulgence of holophilic impulses and proscriptions connected with the "incest barrier" (Œdipus complex).

Readers who will admit that an infant ever refuses the stool "until by a large accumulation it can obtain an increased and more prolonged gratification on evacuation" and readers who are "able to recall from their own experiences that bed-wetting is frequently attended by dreams of acute physical pleasure," should be impressed by a perusal of this book.

The author condemns what he calls the traditional methods of treatment by stern admonition, religious exercises, punishment, etc., and advocates analysis of the subject's mental conflicts, together with sexual enlightenment and sympathetic encouragement. As psycho-analysis is within the reach of comparatively few, this holds out little encouragement for the many who are addicted to the practice.

The book is clearly written and the subject approached without diffidence. The author has troubled to acquaint himself with some of the facts concerning masturbation, but has suffered fancy to walk with them.

The value of the book lies in the fact that new views on subjects of medical interest are always welcome to the profession, if not always accepted. To "parents, teachers and clerics," however, whom many sex writers would include among their readers, it is more likely to bring confusion than enlightenment.

Analytical Department.

PARKE, DAVIS & COMPANY.

THE following report represents the result of an investigation into the process of manufacture and pharmacological properties of another product prepared by Parke, Davis & Company (see THE MEDICAL JOURNAL OF AUSTRALIA, January 28, 1922, page 97; April 22, 1922, page 429; and August 5, 1922, page 160):

"Germicidal Soap."

"Germicidal Soap (2% McClintock)" is a soap said to contain 2% of mercuric iodide. It is recommended as an antiseptic, deodorant and disinfectant for surgical and general medical use. "Germicidal Soap" is prepared from a good quality curd soap, with the addition of mercuric iodide and a blue colouring matter. The soap is mixed with the mercuric iodide and the colouring matter, both in solution, in a milling machine and milled until the mass is thoroughly homogeneous. It is then formed into cakes in dies under pressure. Great care is taken in the manufacture to insure the uniformity of the product.

An examination of the soap obtained at the factory and purchased outside showed that it was very homogeneous in colour and texture, indicating a thorough mixing of the mercuric iodide with the soap. The presence of mercuric iodide was determined, but no quantitative examination was made, as it seemed that a determination of the carbolic co-efficient would be of more use. The carbolic co-efficient of two samples as determined by the Rideal-Walker method was 200. Emulsified in water it killed *Bacillus typhosus* in two minutes in a dilution of 1 in 20,000, corresponding with carbolic acid in a dilution of 1 in 100.

Parke, Davis & Company's "Germicidal Soap" can be confidently recommended as a carefully prepared germicidal soap of high efficiency as an antiseptic.

[1] *Royal London Ophthalmic Hospital Reports*, XIII., 1892, page 104.

[2] "Auto-Erotic Phenomena in Adolescence: An Analytical Study of the Psychology and Psycho-Pathology of Onanism," by K. Menzies, with a Foreword by Dr. Ernest Jones; Second Edition; 1921. London: H. K. Lewis & Company, Limited; Crown 8vo., pp. 100. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, AUGUST 19, 1922.

The Advertising Practitioner.

THE BRITISH MEDICAL ASSOCIATION has been established for the purpose of maintaining the dignity and honour of the medical profession. In commercial circles advertisement is regarded as a permissible means of attracting business. The ethics of commercial advertising are or have become haphazard and ill-defined and are uncontrolled. The advertiser often plays on the credulity of the public and does not allow any code of ethics to interfere with his one object—the sale of his wares. Newspaper managers have the habit of closing their eyes to the abuses of advertising, since every insertion means an increase of income. Every day in every newspaper throughout the Commonwealth there appear advertisements which contain untrue, exaggerated and improper statements. Sometimes, it is true, the unsubstantiated claim is rather implied than expressed and may not be apparent to those responsible for their appearance. Usually, however, both the advertising managers of the newspapers and the advertising agents are aware of the objectionable nature of many of the advertisements which are published. Even when their attention is directed to a particularly glaring offence, the same lying statements continue to appear.

Professional men, as contrasted with commercial men, are required to restrict their advertising to very narrow limits. The medical practitioner has no real need for advertisement. His good work is recognized by his patients and one satisfied patient passes on the word. The best judge of a medical practitioner's worth is his colleague. The public know that the practitioner called in by his colleagues is a good man. Unfortunately, there are always some who are prepared to sacrifice the dignity and honour of their profession for their personal advancement. In every large body of human beings there must be a leaven of individuals whose principles are not of the highest order. Many years ago the General Medical Council in England found it necessary to establish the rule that medical practitioners should not employ the instrument of advertisement for the purpose of attracting patients to themselves. The best authorities in medical ethics deplore even a suspicion of advertisement by medical practitioners and regard all methods other than the display of a name plate as objectionable and wrong. The British Medical Association throughout the British Empire has maintained a high ideal and has striven to banish all open or covert forms of advertising. In the report of the Council of the British Medical Association for the year 1921-1922 the attention of members is drawn to "certain newer journalistic methods" of advertising by members of the medical profession. The Council has approached the General Medical Council with a request to consider the advisability of making a pronouncement on these newer practices. The examples given are the granting of interviews by doctors and the publication of signed letters in the lay press in which the writers hold themselves out to possess or allow themselves to be designated as possessing superior methods of treatment. In other instances the medical practitioner contributes signed articles to the lay press ostensibly for the purpose of imparting useful information, but in reality for the purpose of advertising themselves. The Council deprecates the announcement by a medical practitioner that he is practising as a "specialist" in a particular branch of professional work. The General Medical Council has replied to the effect that it is prepared to deal with specific instances of breaches of the rules concerning advertising by medical practitioners. If a new offence not within the existing "Warning Notice" were brought before it as a specific instance, the matter would receive the Council's careful attention. In other words, the General Medical Council is prepared to extend the rule if it can be shown that subterfuges are being employed whereby the medical practitioner offends against the present rule in spirit but not in letter.

In Australia the Branches of the British Medical Association take a very liberal view of permissible advertising on the part of their members. Members

are permitted to insert notices of change of address, of resumption of practice and of starting practice. Occasionally this permission has been abused, but it must be admitted that the vast majority of practitioners obey the regulations. The "newer methods" of advertising are not restricted to Great Britain. They are well known in Australia. The worst offences are those of well established, prominent doctors, who keep their names before the public through the medium of the morning or the evening newspaper. The Council of the British Medical Association has no hesitation in announcing that many of the signed articles appearing in the lay papers "are not contributions to useful knowledge, but obvious advertisement." It is usually dangerous to attribute motives, but this interpretation at times seems to be justified. Reference is made in the report of the Council to the practice of medical men of taking charge of columns in the daily press in which answers to correspondents on medical matters are printed. The Council regards this as highly detrimental to the public interest and most improper from a professional point of view. While the medical profession can no longer claim that it possesses an exclusive right to medical knowledge and that the layman must be kept in complete ignorance concerning pathological processes or methods of treatment, it is obviously a dangerous expedient to give advice in this way, when the diagnosis is made by the patient, who is not trained as an accurate observer. General information on scientific matters, on the other hand, has a place in the daily paper, but it should be contributed anonymously and the responsibility for accuracy should rest with the editors of the papers. There is no justification for the signing of the articles.

SHIPS' SURGEONS.

THE COMMONWEALTH NAVIGATION ACT, 1912, requires that any foreign-going ship or any Australian-trade ship, whose journey between consecutive ports of call exceeds a certain distance and whose company amounts to one hundred persons or more, shall carry as part of her complement a duly qualified medical practitioner. The penalty attached to a breach of this clause of the Act on the part of the master or owner of the vessel is £100. The provisions of the Act have been brought into operation during the past year. The regulation is a good one. A passenger ship at sea is a self-contained community. The necessities and comforts of life are provided and it is only fitting that provision should be made for the occurrence of illness among passengers or crew. A moment's reflection will enable anyone to realize that, in addition to this, the International Quarantine Regulations make the ship's doctor a necessity. The quarantine medical officer at any port of call can get at the best merely a cursory view of the ship's company. He must rely on obtaining a clean bill of health from the ship's doctor before *pratique* can be granted.

Medical practitioners accept appointments as ships' surgeons for a variety of reasons. Some men prefer the sea, with its healthy freedom; others make one or two trips merely for pleasure, while frequently men have been signed on to the articles of a ship and have drawn their pay of one shilling a month in order to travel cheaply from one part of the world to another. Many people regard the life of a ship's surgeon as a lazy man's existence, while he has been frequently looked down upon and prejudged as incompetent, both by the general public and by his fellow practitioners ashore. This ought not to be. His life is to a certain extent what he makes it and, whether he will or not, it is often no sinecure. The ship's surgeon must be prepared and capable of dealing with any surgical emergency, be it an urgent abdominal lesion or a difficult obstetric complication, and he must have above all a thorough knowledge of infectious diseases. The assistance which he can obtain is limited and his equipment generally leaves much to be desired.

It has long been felt that the remuneration paid to a ship's doctor is seldom proportionate to the services required of him and that his pleasure-seeking *confrère* is rather a hindrance to the betterment of his conditions of service. The Federal Committee of the British Medical Association in Australia endeavoured to secure a degree of uniformity in the conditions of employment and, as far as the Australasian mercantile marine service is concerned, they have been successful. The Honorary Secretary at the instance of the Federal Committee

approached the Australasian Steamship Owners' Federation and suggested that the remuneration paid should be at least £25 per month, with the right to charge certain fees to passengers. The rates proposed were seven shillings and six pence for each consultation to first-class passengers, five shillings to second-class and two shillings and six pence to third-class passengers. The Steamship Owners' Federation agreed to the minimum monthly salary suggested, but demurred on the question of fees. They offered to fix the fee to be charged to both first- and second-class passengers at five shillings and thought that third-class passengers should be treated without fee. After giving the matter full consideration the Federal Committee resolved to accept this proposal.

While it is desirable that similar arrangements should be made in respect of over-seas ships, there are certain difficulties to be overcome. The matter is being considered by the Federal Committee. In any case, the present arrangement with the Australasian Steamship Owners' Federation can be regarded as satisfactory and it is well for medical practitioners to know where they stand in this regard.

THE COLLOIDAL BENZOIN REACTION.

No laboratory procedure of latter times has proved of greater assistance in diagnosis and no test has received more general acknowledgement and use than the Wassermann test. At the same time many practitioners fail to recognize its limitations and unwarranted assumptions are still made when hæmolysis occurs in making the test. The introduction of the ice-box method has made for greater accuracy, but the fact remains that the failure of the patient's serum to fix complement is not conclusive evidence of the absence of syphilitic infection. Consideration of this fact and the complexity of the necessary technique have led to much experimental work in an endeavour to discover simpler and more satisfactory methods.

Sachs and Georgi in 1920 devised the flocculation test and Dreyer and Ward subsequently modified this and labelled their modification the "sigma" test. This reaction was fully discussed in a recent issue of this journal. The greatest difficulty occurs in the diagnosis of cerebro-spinal syphilis. It is not uncommon to find patients suffering from *tabes dorsalis* or general paralysis of the insane whose blood serum fails to yield any reaction to the Wassermann test. And, further, syphilitic lesions of the nervous system may be not only active but progressive in patients whose blood serum under anti-syphilitic treatment fails to yield a reaction. This is well exemplified in the so-called nerve relapses or *neuro-récidives* which are occasionally noticed after full doses of salvarsan or similar arsenical preparation. The cerebro-spinal fluid may yield a reaction to the Wassermann test when the blood serum does not do so. The changes in the cerebro-spinal fluid which cause the reaction, are of a similar nature to changes which apparently endow it with the property of precipitating certain colloids. Lange in 1913 elaborated what is known as the colloidal gold test. In this test a specially prepared solution of chloride of gold is added to a series of dilutions of cerebro-spinal fluid in sodium chloride solution. The reaction consists in the precipitation of the gold and this is indicated by a gradual change in colour, leading to complete decolorization. The point at which the maximum decolorization occurs is said to distinguish syphilitic from the so-called para-syphilitic conditions. Cruickshank held that the precipitation was due to the action of the globulins in the cerebro-spinal fluid and that the albumin fraction acted in the opposite direction by exerting a protective influence on the suspension of the test solution. The test has been proved to be of definite value. The drawbacks to its use are the delicate nature of the technique that is necessary and the difficulty that is experienced in preparing satisfactory solutions of the colloidal gold.

In 1920 Gullain, Guy-Laroche and Lechelle showed that a similar reaction took place if a colloidal solution of gum benzoin were substituted for chloride of gold. Dr. J. Henry Dible has recently investigated this test and published the result of his observations on its use with fifty-one samples of cerebro-spinal fluid.[1] The technique used by Dr. Dible was not complicated. A progressive series of dilutions of cerebro-spinal fluid in saline solution was prepared. The first tube contained three parts of cerebro-spinal fluid to one of saline solution, the second one in two and so on up to the fifteenth, which contained one in 16.384. An equal quantity of freshly prepared colloidal suspension of gum benzoin was added to each tube. The tubes were allowed to remain at room temperature. If precipitation occurred, its appearance was rapid and in the form of flocculation which formed a sediment at the bottom of the tube. The results were read after a few hours. The originators of the test stated that no precipitation occurred in normal cerebro-spinal fluid and in that in late syphilitic disease of the nervous system a precipitate was produced in the more concentrated solutions, this precipitate gradually disappearing as the higher dilutions were reached. Dr. Dible, however, found that a certain amount of precipitation always occurred with normal cerebro-spinal fluid. This appeared in the fifth or sixth tube of the series and was present in a short zone over four or five tubes. No explanation is attempted as to why this difference should occur. The precipitation, when it occurred, spread over a wide range, beginning generally in the first tube and extending to very large dilutions in the tenth or twelfth tube.

Dr. Dible applied the test to fifty-one samples of

[1] *The Lancet*, June 3, 1922.

cerebro-spinal fluid. With seventeen samples of fluid yielding a reaction to the Wassermann test, the benzoin test led to precipitation in sixteen and in one the result was indefinite. With thirty-four fluids which failed to yield a reaction to the Wassermann test, no reaction occurred with the benzoin test in twenty-five, precipitation resulted in two, while the result was indefinite in seven. The series of experiments is too small to draw any definite conclusions as to the comparative value of the tests. It would appear, however, that the results coincide closely with those obtained in the Wassermann test.

In discussing the nature of the reaction, Dr. Dible states that he had sought to explain the precipitation as being due to increased amounts of electrolytes in the fluid acting on the electro-negative suspension of colloidal benzoin. He tested this hypothesis and found that it could not be substantiated. In view of the fact that the protein content is generally increased in fluids yielding a reaction, he is inclined to accept the view expressed by Cruickshank in regard to the colloidal gold test, although he states he made no globulin estimations.

It would appear that this test, together with the colloidal gold and the flocculation tests, is likely to prove of value and to take its place as an additional means of diagnosing syphilis, especially that of the nervous system.

THE USE OF DIGITALIS IN PNEUMONIA.

TREATMENT of a disease may be undertaken from several standpoints. In the first place the causal organism may be directly attacked with the object of staying the disease process, either by destruction of the organism or by the inhibition and nullification of its noxious products. Treatment may be symptomatic and here the measures undertaken are directed at combating the effects of the infection as evidenced in the patient's condition. The cure of the disease is left to the patient's resistance. Lastly, treatment may be empirical. By this is meant that measures are employed speculatively, the reason for their use being the fact that they have proved to be beneficial in similar conditions.

In dealing with pneumonia, all these methods have been used at various times. Many efforts have been made to apply forms of treatment that were supposed in turn to be of universal benefit in the disease. The open-air treatment of pneumonia has had its staunch adherents and much was hoped from the use of serum. Clinicians have had to recognize, however, that the field of usefulness of these methods, as of others, is limited. At the same time, the routine adoption of symptomatic treatment, where there are reasonable grounds for hoping to achieve success, should not be rejected until a fair trial of the method has been undertaken.

Brooks and Carroll studied the question of the management and care of the heart in pneumonia and of the use of digitalis in this connexion. They dealt with patients suffering chiefly from influenzal broncho-pneumonia in the American Expeditionary Force in France during the war and claimed beneficial results from the use of digitalis. Death in pneumonia is often due to cardiac failure. That an undue strain is placed upon the heart in a disease in which the lungs are extensively involved, is obvious and it would naturally be expected that in such a condition as pneumonia a certain amount of degeneration, with resultant impairment of function, would occur in the delicately constituted myocardium. The recuperative power of the heart is great, as evidenced in the myocardium, which is so frequently the seat of much damage by the bacillary toxins in diphtheria. The heart, moreover, recovers wonderfully in enteric fever, when the exhausting conditions of the disease and secondary infections undoubtedly play an important part. If any form of treatment can be devised that will assist the heart in times of stress, something of value will be accomplished.

Dr. Willard J. Stone has studied this question and has recently published the results of his investigations.[1] Dr. Stone's communication deals with a series of 1,205 cases of lobar and broncho-pneumonia, observed from October, 1917, to December, 1918, together with 259 *post mortem* examinations. In 283, or 23%, septic complications occurred in the form of empyema, pericarditis, sub-sternal pus pockets, abscess of the lung, sub-diaphragmatic abscess and peritonitis. Dr. Stone does not give much detail of these complications, but states that in a series of operations for empyema performed during this period, streptococci, chiefly *Streptococcus hæmolyticus*, were responsible in 72% and pneumococci in 27%. In eighty-nine autopsies following lobar pneumonia the heart had normal macroscopical appearances in 57.3%. Dilatation of the right ventricle was present in 39.4% and of the left in 12.4%. Parenchymatous degeneration was the principal microscopical change found on examination of the heart muscle of eighteen patients out of a series of thirty-four who died of the disease. Digitalis therapy was used as a routine procedure in all patients treated from January 16 till the end of the year. The total dosage was given to patients during the first thirty-six hours after their admission to hospital. The special tincture of digitalis used was standardized by the cat method of Hatcher. Of this tincture a dosage of 0.3 cubic centimetre per kilogram of body weight was used. Thus a patient weighing seventy kilograms received twenty-one cubic centimetres (about six fluid drachms). As a rule, no definite slowing of the pulse rate was noticed. Dr. Stone does not publish any records of pulse rates, pulse tracings or cardiograms showing the heart's action before or after treatment with digitalis. Occasional nausea was produced. Partial and temporary heart block occurred only once and coupled beats and frequent premature contractions were not common. These symptoms usually subsided after the hypodermic injection of 0.6 milligramme (one one-hundredth of a grain) of atropine. A transient rise in systolic blood pressure was commonly noticed and diuresis was the most constant evidence of absorption and effect. If further dosage became necessary after four or five days because of a dilated heart, the earlier doses were supplemented by intravenous injection of *digipuratum*, by digi-

[1] *The American Journal of the Medical Sciences*, May, 1922.

talone or by the oral use of Nativelle's crystallized digitaline. Of 279 patients to whom digitalis was not given as a routine treatment, the mortality was 41.9% and of 926 patients treated by digitalis the mortality was 18.2%. No account is given of the treatment that was adopted when digitalis was not used. Dr. Stone also states that antiserum was used in all cases of pneumonia due to Type I. pneumococcus. He does not state the percentage number in which this occurred. No conclusion can thus be formed on account of the possible combination of digitalis and serum or the separate use of either. Dr. Stone concludes that digitalis, if used at all, must be used early in the disease and that small doses of the tincture three or four times a day are useless. He shows that in this way it would take twenty days to give an amount similar to that administered by him in thirty-six hours.

The possibility of beneficial results are manifested by this work, but Dr. Stone's figures must be accepted with reserve. His statement of the case leaves something to be desired in regard to the control of his observations.

FOCAL EMBOLIC GLOMERULO-NEPHRITIS.

PATHOLOGISTS formerly endeavoured to study the special lesions found in the several organs, more or less dissociated from the processes which laid the foundation for these lesions. They were usually satisfied to register an association between a disease process and a pathological change in certain organs and to leave the mechanism of the production of the special lesions unexplored. During the last few years the unsatisfactory nature of this form of study has been recognized and endeavours have been made to fathom more exactly the ætiological relationship between a general affection and a local manifestation. For example, the pathology of scarlatinal nephritis is not exhausted when the histology of the renal changes are recorded and the successive stages followed from the earliest involvement to the most extensive inflammatory lesions. In the early stages of a glomerular inflammation the changes appear to affect all the glomeruli in many contiguous tufts at one time. The lesions appear in response to the invasion of the organ by a bacterium or by some chemical product of disturbed metabolism and the extent does not vary greatly in different parts of the affected tissue. Within recent years a peculiar form of glomerulo-nephritis has been discovered in which this equal distribution is not maintained. In 1910 Löhlein noted that the kidney of some persons dead of ulcerative or subacute bacterial endocarditis were affected in this way. A few loops in a tuft are found to be changed, while contiguous loops remain intact. He studied this condition closely and arrived at the conclusion that the process was an embolic one and that it was manifested as a definite nephritis during life. Other pathologists investigated this characteristic focal embolic glomerulo-nephritis and brought forward evidence in support of the contention that it should be regarded as a distinct pathological entity. The first change in the glomerular loop appears to be a swelling of the endothelial cells. The cells pass through a regular sequence of destructive changes until their remains are represented merely by a hyaline mass. The nucleus disappears at an early stage. At times the adjoining cells undergo proliferation. The affected loop may finally become adherent to the neighbouring part of Bowman's capsule. A crescentic appearance has been noted in the glomerulus, caused by this sclerosis and fusion of some of the contained loops. A curious feature of the process is the variability of its distribution. At times few tufts are involved and the condition may be missed unless a very careful search is made. In other instances nearly all the glomeruli are affected and but little functionating kidney remains. The tubules leading from the involved glomeruli usually contain red blood corpuscles.

Dr. Margaret Warwick has collected information concerning this condition in ten persons who had died of ulcerative or subacute bacterial endocarditis.[1] She has added the information gained to that previously collected by other observers. Baehr, an American pathologist, had sought in 1912 to establish the fact that the minute emboli leading to the lesions were composed exclusively of masses of bacteria. In the majority of instances the infecting organism is a *Streptococcus viridans* of low-grade virulence. He succeeded in staining the bacteria *in situ*, but other workers have failed to achieve this. It is suggested that the actual lesions may be caused by chemical agents originating from the bacterial action on the tissues. It is certainly a significant fact that both the cardiac lesion and the renal changes may exist in a healed condition, while the clinical manifestations are still progressing. In addition to the changes in these two organs, enlargement of the spleen and infarctions of the spleen, liver and kidney are commonly observed. During life the association of an endocardial affection with hæmaturia, albuminuria and the presence of casts in the urine should arouse a suspicion that a streptococcus of low-grade virulence had given rise to an infection of the endocardium and subsequently to embolic processes affecting the glomeruli. All attempts to correlate the site of the vegetations in the heart with the tendency to the renal complication have proved unavailing. In fact, focal embolic glomerulo-nephritis has been noted in a person whose endocardium has been free from infection. Dr. Warwick is of opinion that the peculiar lesions in the kidneys may be caused by almost any bacterium of low-grade virulence and that it does not depend on a specific micro-organism. She bases this assumption on the finding of *Streptococcus viridans* in the majority of the kidneys and *Streptococcus hæmolyticus* in a few. It is unlikely that her conclusion that the lesions may be entirely mechanical is sound, in view of the nature of the changes. It appears, however, that the essential lesions may lead to a lowering of the resistance of the whole organ. At times a chronic diffuse nephritis may be grafted on to the affected kidney. Practitioners who have an opportunity of studying the *post mortem* findings in persons who have died of subacute bacterial endocarditis, should pay special attention to the histology and bacteriology of the kidneys.

[1] *The Journal of Laboratory and Clinical Medicine*, June, 1922.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Keratomalacia in Southern India.

R. E. WRIGHT (*British Journal of Ophthalmology*, April, 1922) compares the common clinical condition, keratomalacia, so often treated in the Madras Eye Hospital, with the experimentally produced effect in animals, the result of deprivation of fat-soluble vitamin and designated by the same name. Experimental keratomalacia or xerophthalmia is produced in white rats in about forty days by excluding fat-soluble A from the diet. The eyes become swollen so badly that they are opened with difficulty or not at all. The cornea becomes inflamed and, unless the missing essential dietary is supplied, blindness speedily results. Complete recoveries were obtained within two weeks of starting to administer a diet containing 5% of butter fat, provided the animals had not been previously blind and normal eyelids could be restored, even if the cornea were destroyed. The clinical variety is seen most often during periods of scarcity and famine. Children under five years of age are most commonly affected, but it may occur in older children or in adults, especially in those suffering from chronic disease of the intestine. The principal cause is the lack of some essential substances in the dietary or, if such are present, to an inability to assimilate them. A frequent premonitory sign is night blindness and the disease begins by a stiffening of the triangular area of conjunctiva on each side of the cornea. Both eyes are affected. The hair appears dry and the skin roughened. The triangular patches are darker than the surrounding conjunctiva and soon show a white formation like soap lather on their surfaces. A faint grey haze appears between the centre and periphery of the cornea and spreads all over its surface. An hypopyon may develope, but there is no vascular reaction. The tissues of the cornea appear to melt away and slough off, the ultimate end being *phthisis bulbi* or the death of the patient. The local treatment is mostly cleanliness, warmth and protection. Atropine is generally harmful. Constitutional treatment is most important. Castor oil should be given for the diarrhœa which is usually present and cod liver oil is the essential remedy. A mixture of chlorides of calcium, sodium and ammonium is valuable. Fruit juice and thyreoid extract may be given with advantage. A definite experiment of adding fat-soluble A vitamin to the diet was made in 1920. A fresh mixture was prepared daily, consisting of equal parts of yolk of egg, fruit juice, butter (ghee) and raw meat juice. Adults received 240 cubic centimetres (eight ounces), babies, 60 cubic centimetres (two ounces) *per diem*. No beneficial result was obtained. The bulk of the poor population are chronically underfed; they are in what McCollum calls the "twilight zone." Their diet consists of rice swelled with pepper water, or a thin, vegetable curry with few, if any, green leafy vegetables. Milk, meat, eggs and butter are seldom taken. The clinical condition, therefore, does not correspond to the experimental, nor does it respond in the same way to the administration of the required diet. The problem of prevention is an economic and financial one.

Prechiasmal Intracranial Tumour of the Optic Nerves.

W. E. DANDY (*American Journal of Ophthalmology*, March, 1922) reports two cases of tumour of the optic nerve between the optic foramen and the chiasma. The diagnosis is difficult owing to the absence of characteristic symptoms; there is no proptosis, no hemianopsia, no X-ray appearance of erosion of the *sella turcica*, no optic neuritis, but only failing vision and atrophic discs. The first patient was a girl, aged thirteen years. Since her seventh year she had had several vomiting attacks, with diplopia and failing sight. On examination the right eye was found to be totally blind and the left vision $^{5}/_{200}$ dioptre. The discs were atrophic. Despite the absence of headache, the transient bilateral extraocular palsies accompanying the spells of vomiting and the slow, progressive blindness seemed to indicate a tumour. Only a pineal tumour or one in the region of the sphenoidal fissure could produce bilateral ptosis and extraocular palsies. The pineal region was excluded by the absence of optic neuritis and high intracranial pressure. At the operation two independent tumours were found surrounding each optic nerve at the foramen, 1.5 centimetres in diameter. and extending into the orbit for about 1.5 centimetres. The tumours were endotheliomata arising from the dural sheath, with features of a psammoma. The growth on the right optic nerve was totally removed, including the orbital portion, through a resection of the orbital roof. The left tumour could be only partially removed, but vision was restored to $^{20}/_{200}$ dioptre. The second patient was a boy of eight years, blind in the right eye with $^{20}/_{200}$ dioptre vision in the left. Both discs were pale. No headache, vomiting or palsies were present. A right craniotomy over the frontal region was performed. The right optic nerve was normal as far as could be seen, but a tumour the size of a cherry was found beneath the left optic nerve, which was also constricted by a tight band of fibrous tissue between the anterior clinoid process and the optic foramen. This band was excised, but the tumour was too far distant for removal. The tumour did not seem to enter the optic foramen. The result was a distinct improvement in the sight of the left eye to $^{20}/_{20}$ dioptre and to $^{20}/_{70}$ dioptre in the right. Many intra-orbital tumours extend into the cranial chamber; orbital operation does not therefore meet the case. When such an extension exists, only an intracranial operation can save life or vision.

Differential Pupilloscopy.

O. BARKAN (*Archives of Ophthalmology*, January, 1922) describes Hess's method of measuring the pupillary reaction to light. The eye is viewed through a telescope of eight magnifications. The light thrown upon the eye passes through a grey glass or through an arrangement of prisms whereby its intensity may be varied and measured by the observer. By swinging these up and down the eye will be successfully illuminated with lights of equal or varying intensity and the contraction of the pupil may be studied. The least difference of light to excite a reaction is called the motor discriminative acuity. This occurs normally when the light intensities vary as 95 to 100. It is usually expressed as a quotient, 0.9. Diminution of motor discriminative acuity to 0.84 is pathological. The same instrument is used to test subjectively the smallest light difference, called the visual discriminative acuity. The author goes on to describe the pupillary reflex arc and shows that in dioptric lesions the motor and visual discriminative acuities are both normal. In lesions of the intercalated portion (the unknown region where the pupillary fibres branch off from the optic tract to reach the third nucleus) the motor discriminative acuity is diminished while the visual discriminative acuity is intact, whereas in cortical lesions the motor discriminative acuity is normal and the visual discriminative aculty is impaired. Comparison of the direct with the consensual reaction gives valuable diagnostic aid in differentiating between a sensory lesion (optic nerve, retina) and a lesion in other parts of the reflex arc. The pupilloscope gives exact numerical values for the consensual and direct reaction. It is found that in a lesion of the intercalater path (Argyll-Robertson pupil) the direct and consensual motor discriminative acuities of the same eye are equal, whereas in a receptor or fibre lesion the consensual motor discriminative acuity of one eye is equal to the direct motor discriminative acuity of the other eye.

Episcleritis: A New Method of Approach.

H. L. SINSKEY, M. B. LEVIN AND B. SACKS (*Archives of Ophthalmology*, November, 1921) report the case of a woman of thirty-six who suffered from attacks of inflammation of the left eye, with the formation of an episcleral nodule. The condition was diagnosed as episcleritis and treated for about two years by four different oculists. The usual methods of examination were followed. For instance, she had teeth removed for pyorrhœa and the tonsils were also excised. Except for short intermissions, she was no better. The present authors then made elaborate experiments in dietary and demonstrated that the omission of carbo-hydrate, in the form of cereals, potatoes, bread and sugar, and the reduction of fat caused a disappearance of the symptoms. Ingestion of these things caused a relapse.

LARYNGOLOGY AND OTOLOGY.

The Mechanism of the Cochlea.

ACCEPTING provisionally the theory of hearing of Helmholtz, *viz.*, that of sympathetic resonance, GEORGE WILKINSON (*Journal of Laryngology and Otology*, December, 1921) attempts to show that the tensile strain on the various sectors of the basilar membrane may be approximately calculated. The factors determining the period of vibration of any vibratory body are the length of the vibrating segment, its tension and its mass. Helmholtz found that the fibres of the basilar membrane became progressively longer from the base to the apex of the cochlea and Gray showed that the tension of these fibres varied progressively in the same sense as the variations in length, *i.e.*, the shorter fibres were the tenser and the longer the looser. Every tense string has a definite periodicity and is formula is:

$$n = \frac{1}{2l} \sqrt{\frac{t}{m}}$$

The range of values of n (the limit of audible tones) and l (the varying breadth of the basal membrane) are known, but that of the tension and mass is not. The mass of unit length of the vibrating element is represented by m, which Wilkinson says may be regarded as a double column of fluid, the base of which is equal in area to the surface of a sector of the basilar membrane at any particular level and the length to the sum of the distances of the sector from the round and oval windows. As m is a mass of unit volume, it will not vary with the variations of transverse breadth of the basilar membrane. For sectors of the same width m progressively increases from base to apex of the cochlea and the load is greater on the sectors that are longest and least tense. The formula when applied to the basilar membrane becomes

$$n = \frac{1}{2l} \sqrt{\frac{t}{db}},$$

d being the sum of the distances of the sector from the round and oval windows and b the width of the sector. If this formula be accepted as correct, it becomes possible to calculate approximately the maximum and minimum values of t. Employing Keith's measurements, the author shows that the tension on a strip one millimetre broad at the proximal end of the basement membrane would be 18.6 grammes and at the distal end 4.6 milligrammes. Wilkinson, after experimenting on the breaking strain of various animal fibres, concluded that the basilar membrane could readily withstand the maximum tension calculated by the above formula.

Intrinsic Cancer of the Larynx.

INTRINSIC cancer of the larynx originates on the vocal cords or in the sub-glottic area. In fifty patients examined indirectly as well as directly (by largyno-fissure) by ST. CLAIR THOMSON (*Journal of Laryngology and Otology*, October, 1921) it was not found in the posterior commissure nor originating from the ventricular bands or the ventricle. It may arise from any part of the cord, but is more common in the central portion or anterior half than in the posterior area of the larynx. An epithelioma may remain for a long time limited to the cord affected and the adjoining side of the larynx, but it may cross the anterior commissure and in later stages invade the arytenoid and the area to the outer side of it. The inner surface of the cord may be affected primarily or by extension. The sub-glottic area may be invaded by a growth originating in the cord, or the growth may commence in the sub-glottic tissues. A sub-glottic cancer is much more common in the anterior than in the posterior half of the larynx. As regards prognosis, the superficial or projecting tumours of limited extent are the most favourable. Those situated in the middle third or anterior half of the cord are more promising than those invading the anterior commissure or the arytenoid region. Growths imbedded in the cord or extending into it below an intact mucosa are not so favourable. An epithelioma extending along the inner margin of the cord is still less favourable. Sub-glottic cancers are very unpromising as regards lasting cure by laryngo-fissure. They are frequently associated with impaired mobility or complete fixation of the cord. In every case, however limited the growth, the entire vocal cord should be excised from the anterior commissure up to and including the vocal processes of the arytenoid. The growth, with as wide a margin as possible of apparently healthy tissue all around it, should be removed in one mass. The incision should go down to the lower edge of the sub-glottic area; above it should pass through the healthy ventricular band; externally it must include the perichondrium lining the thyreoid ala. To facilitate this the thyreoid ala should be removed.

Residual Hearing.

MAX. A. GOLDSTEIN (*Journal of Laryngology and Otology*, October, 1921) recommends a careful study of the speech and hearing centres and their relation to the various types of serious deafness as an essential preliminary to the effective investigation of the nature of deafness in young children and its possible alleviation. There may be revealed an uneducated or latent auditory word centre, an imperfectly developed Wernicke's area, lack of correlation between Broca's and Wernicke's areas or some other undeveloped or imperfectly stimulated speech or hearing centre. The clinical types into which deafness is classified by Goldstein for this study are: (*i.*) Lymphatic, the end result of which is impairment of function of the conducting apparatus. (*ii.*) Exanthematous, in which suppurations are of a specially destructive type, leading to necrosis, erosions and permanent ankylosis or bony changes. These invasions may be accompanied by a toxic selective attack on a part of the labyrinth or meninges. The cochlear cells may be intermittently and irregularly attacked, producing definitely recognized and still functioning tone-islands throughout the cochlear scale. (*iii.*) Central, in which there is a sudden attack on the auditory central organ and total deafness, as occurs in epidemic meningitis, poliomyelitis and exanthemata with intense febrile reaction. (*iv.*) Oto-sclerotic, occurring more often in adult life and hence not taking so large a part in the problem of residual hearing and its development. (*v.*) Congenital, ascribed largely to matings of the congenitally deaf and to consanguineous marriages. (*vi.*) Hereditary, due to the transmission of a degenerative element, as in rickets and syphilis. (*vii.*) Benign, in which no permanent secondary changes have followed local affections of the nose or post-nasal areas. The presence of any residual hearing in any of the above conditions should be ascertained and cultivated.

Nasal Sinusitis in Children.

F. J. CLEMINSON (*Journal of Laryngology and Otology*, November, 1921) has recorded his observations in a study of eighty-five cases of nasal sinusitis in children. He notes that the symptoms, in descending order of frequency, are as follows: coryza, nasal obstruction, constant colds, deafness, chronic cough, frontal or supra-orbital headache, mental dulness and lethargy, sometimes epistaxis and pains in the face, teeth or joints. Similarly the signs are: pus or mucopus or a clear, watery fluid in one or both sides of the nose, thickening, reddening and excoriation of the nostrils, congestion of the turbinals and sometimes a traumatic septal deflection. Trans-illumination, radiography and exploratory puncture assist in diagnosis. In treatment one puncture, followed by irrigation, may be all that is required to effect a cure or several irrigations may be necessary. In addition, an alkaline nasal douche, followed by an oily application to the interior of the nares, may be used. The author is in favour of removal of the tonsil and adenoid vegetations as part of the treatment. Occasionally the removal of the anterior end of the middle turbinal is required to secure good drainage.

Otitis in Infants.

ROBERT LEROUX (*La Presse Médicale*, December 17, 1921) affirms that otitis exists in a latent state in all newly born children. Anatomical and pathological conditions explain its frequency. The ear can become infected during birth with the first inspiration. The ears, like the eyes, should be attended to from birth and the nasal fossæ should at this time be disinfected. In every illness the ear should be examined, especially when unexplained symptoms are present. A paracentesis should be performed if the ear condition is doubtful.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Queensland Branch of the British Medical Association was held at the Brisbane General Hospital on June 2, 1922, DR. G. P. DIXON, C.B.E., the President, in the chair.

THE PRESIDENT, after the formal business had been transacted, vacated the chair in favour of DR. EUSTACE RUSSELL, the President of the Brisbane Hospital Clinical Society.

Skin Eruptions.

DR. J. G. AVERY presented a married woman, aged forty-two years, who complained of a rash on the face and legs. She had one child in apparently good health. The rash had developed fourteen days previously and was papulo-macular in type. No primary syphilitic lesion could be discovered. The patient's serum did not fix complement in the Wassermann test.

He also showed a female, aged fifty-five years, with a papular rash on her palms. The rash was typical and a strong reaction had been obtained to the Wassermann test.

The third patient was a female, aged twenty years, who had a papular rash on her legs and forearms. Dr. Avery regarded this rash as a psoriaform seborrhœa of unusual type.

DR. A. GRAHAM BUTLER, D.S.O., and DR. V. MCDOWALL expressed the opinion that the rash was psoriasis.

DR. J. L. SPENCE thought that the condition was an ordinary seborrhœa under too intense treatment. He pointed out that the patient had distinct *pityriasis capitis*.

DR. EUSTACE RUSSELL concurred with Dr. Spence in his diagnosis.

Aerophagy.

DR. EUSTACE RUSSELL showed a returned soldier suffering from aerophage associated with a history of exposure to poison gas. At first the condition had been regarded as an organic abdominal affection with flatulent dyspepsia. The patient had proved refractory to psycho-analysis, partly on account of alcoholic excess. Weir-Mitchell treatment had also proved valueless. Great improvement had been noted after gastric lavage, but the condition had recurred as a result of the patient's alcoholic habits. Dr. Russell stated that the condition had first been described in detail by Wyllie, of Edinburgh. The man was a "wind sucker" and not a "gulper." The condition was at times associated with gross cardiac disease, such as *angina pectoris*, while in other instances it was a pure neurosis. Dr. Russell considered that the patient's condition was a neurosis.

DR. S. F. MCDONALD stated that he had seen two patients with this condition at the Repatriation Department. The condition had proved very difficult to treat by psycho-analysis. It was difficult to ascertain the cause, especially if the condition were complicated by hereditary defects and by alcoholism. Muscle re-education was at times of benefit. He had seen another patient with this affection in hospital. The diagnosis in that patient had been Hirschsprung's disease. A skiagram had revealed an enormous dilatation of the stomach.

DR. A. V. MEEHAN had seen Dr. Russell's patient at Rosemount, when Colonel Strangman had given him a long course of muscle relaxation of the neck. The benefit had been temporary.

DR. RUSSELL in reply said that he had been asked to administer an anæsthetic to a patient who was said to be suffering from gastroptosis. Just before the second stage of anæsthesia he had noticed that the patient was a wind swallower and that the stomach was becoming distended. He had warned the surgeon, but the latter had proceeded to perform the operation of gastropexy. The condition of acute dilatation of the stomach had developed and the patient had died a few hours later.

Sternberg's Disease.

DR. EUSTACE RUSSELL read notes of a condition diagnosed as probably Sternberg's disease.

A discussion followed.

Calculus Following Prostatectomy.

DR. A. G. ANDERSON presented a male patient, aged seventy-eight years, on whom the operation of prostatectomy had been performed in 1917. The patient had remained in good health until July, 1921, when frequency and difficulty of micturition had re-appeared. There had been no signs of cystitis and the urine had not contained blood. At first no abnormal appearance had been detected by cystoscopy. Later a stone had been felt 1.25 centimetres external to the orifice of the bladder. It had probably been caused by phosphatic deposits in the bed of the prostate. There had also been an ulcer beyond the intertrigonic band. The calculus was clearly seen in the skiagram exhibited.

Cardio-Spasm.

DR. R. GRAHAM BROWN showed a patient suffering from *tabes dorsalis* with symptoms of cardio-spasm. The patient was seventy-seven years of age. He had had difficulty in swallowing for four days. This had been followed by dysphagia. A skiagram revealed a stricture of the cardiac end of the œsophagus. The condition had been diagnosed after the passage of the œsophagoscope. The knee jerks were absent and Romberg's sign was elicited.

Diathermy.

DR. A. T. H. NISBET demonstrated the end result of the treatment of a rodent ulcer by diathermy. The patient was a man, aged forty-three years. The ulcer had been of considerable size. In addition, the patient had had an epithelioma of the lip which had been excised, together with the lymphatic glands of the area.

DR. R. GRAHAM BROWN called attention to a man, aged sixty-eight years, who was suffering from carcinoma of the palate. Very considerable improvement had occurred after two applications of diathermy. This patient had also had carcinoma of the larynx. The larynx and the regional lymphatic glands had been excised.

Fractured Skull.

DR. C. J. WEEDON presented a man with a history of having been hit on the head by a stone during the process of blasting. He had not lost consciousness, but had vomited and had complained of headache. There had been a lacerated wound of the skull 7.5 centimetres above the eyebrows and a depressed fracture of the frontal bone. The skull had been trephined and, as no fissuring of the surrounding bone had been discovered, the displaced fragment had been replaced. Sepsis had occurred and a small fragment of bone had been removed. At the time of the demonstration there was a depressed scar. No pulsation was seen and the man felt no ill-effects.

Chronic Ulcer of the Leg.

DR. C. J. WEEDON showed a man with a chronic ulcer of the leg of ten years' duration. The ulcer had penetrated to the bone; the ankle was enlarged and the foot everted. A skiagram revealed an old-standing ankylosis of the ankle joint and a hollowing of the lower end of the tibia with almost complete disappearance of the bone. The condition resembled an osteo-myelitis or a neoplasm.

Hour-Glass Contraction of the Stomach.

DR. C. J. WEEDON read the notes concerning a patient with hour-glass contraction of the stomach. It had been recorded that the patient had vomited and had had pain in the epigastric and right umbilical regions. The condition had been demonstrated at an examination after a bismuth meal. An operation had been performed and a definite constriction had been found. As the patient had taken the anæsthetic badly, Dr. Weedon had been able only to perform a posterior gastro-enterostomy to the upper loculus. The patient had made a rapid recovery. No trace of malignant involvement of the organ had been detected.

Exostosis of the Spine.

DR. WEEDON'S last demonstration was of special interest in connexion with the provisions of the *Workers' Compensation Act*. A man had lifted a case and later had complained of severe pain in his back. A skiagram of the

spine revealed several exostoses. His blood pressure had been 210 millimetres of mercury. His urine had contained some albumin. Acute pain had been felt down the left leg, associated with an area of numbness over the left shin. The left hip had been examined, but nothing abnormal had been found. There was an exostosis on the right side which apparently was not causing any symptoms. The pain corresponded to the distribution of the anterior crural nerve.

Skiagrams.

DR. V. MCDOWALL exhibited a series of skiagrams. They included shadow pictures of a radius injured by a gun shot and treated by bone grafting. The end result was good, notwithstanding the occurrence of severe sepsis. The next was the picture of an aortic aneurysm; the third was a sarcoma of the femur with secondary deposits in the chest. Other pictures shown illustrated pneumokoniosis and pulmonary tuberculosis, tuberculosis of the elbow joint and miliary tuberculosis of the lungs and fracture of the neck of the scapulla.

DR. R. GRAHAM BROWN presented a series of X-ray negatives illustrating follicular odontomes and dental cysts.

A MEETING of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on July 7, 1922, DR. G. P. DIXON, C.B.E., The President, in the chair.

Otitis Media.

DR. R. GRAHAM BROWN exhibited a skiagram of the skull of a patient in whom *otitis media* had followed an attack of influenza. He stated that there was an epidemic of influenza characterized by suppuration of the middle ear. The onset was usually sudden and violent. Paracentesis was not as effective as in ordinary suppurative lesions of the middle ear and at times had to be repeated. The pus seemed to be retained in the attic.

Carcinoma of the Palate.

DR. GRAHAM BROWN also showed three patients with inoperable carcinoma of the palate and throat. The condition was being treated with diathermy with promising results.

Petit Mal.

DR. A. C. F. HALFORD showed a girl, aged nine years, who was suffering from *petit mal*. Her father had stated that she ran at times to a couch or bed or to her parents, calling out that she was going to have a "turn." The attacks were of varying types. Sometimes they were slight convulsive twitchings; at other times the convulsions were severe. She clutched her right hand as if she felt the attack starting in this situation. She appeared to struggle against the attacks and often swallowed "a lump in her throat." The attacks lasted for a varying time; they were repeated for twenty or thirty minutes. At times two or three days elapsed between the attacks. They usually occurred in the early morning. During a severe attack there was loss of power of the right side and interference of speech. Before the onset she complained of pain in both temples and a "pulsating" pain in the left hypochondrium. She had had convulsions as an infant after a gastric attack. The epileptic condition had started at the age of two years and three months. The parents had experienced difficulty in making her micturate or defæcate. The birth had been normal and no hereditary influence had been detected in the family history. Examination of the eyes revealed a slight degree of hypermetropic astigmatism. No abnormal difference in ratio between the size of the cerebrum and that of the cerebellum had been detected by X-ray examination. There was an unusual projection from the inner table in the region of the upper frontal area. The child was being treated with bromides, but no lasting benefit had resulted. Dr. Halford sought the views of the members concerning the most promising lines of treatment.

DR. A. JEFFERIS TURNER said that there was no focal lesion. The transient weakening of the right side was due to the left hemisphere working more actively than the right hemisphere. The prognosis was bad because of the slightness of the attacks and their frequence. In addition, no benefit had resulted from bromide treatment. He regarded it as a very bad sign that the child had already been under treatment for seven years. Indiscretions in diet would render the attacks more frequent. He recommended a combination of belladonna and bromides and would be inclined to try borates as well.

DR. S. F. MCDONALD considered that the outlook was bad. There was not only a definite epileptic basis, but some functional, hysterical factors. Some of the attacks could be controlled by will power. Segregating the child would have the effect of increasing her conviction that her affection was incurable. As no epileptic colony was available, it would be better to allow her to mix with other children. He did not have great faith in bromides; in this instance he would prefer to give bromides in combination with borates. He had found that "Luminal" in doses of 0.06 gramme (one grain) given at night time was beneficial. Patients taking "Luminal" tolerated bromides better.

Ovarian Cyst.

DR. HALFORD also exhibited a specimen of an ovarian cyst and pyosalpinx. The patient had come to him with an acute suppurative condition in the pelvis. The abscess had been opened from the vagina, but no improvement had manifested itself within four days. He had then opened the abdomen and had found a large ovarian cyst. Behind it and extending into the right hypochondrial region was a large pyosalpinx of the right tube. He had apparently drained a pyosalpinx of the left tube through the vagina.

Ring Sarcoma of the Ciliary Body.

DR. J. LOCKHART GIBSON read notes of a case of ring sarcoma of the ciliary body and exhibited a specimen (see page 215).

Convulsions in Children.

DR. A. JEFFERIS TURNER read a paper entitled "Convulsions in Children" (see page 203).

DR. C. A. THELANDER stated that many mothers held the opinion that bananas and grape skins caused convulsions. Convulsions not epileptic in nature were toxic manifestations and were accompanied by pyrexia. He held the opinion that they should not force food into the stomachs of these children. The slighter the attacks, the graver was the prognosis.

DR. W. CROSSE had seen several children recently with obscure fever, fits and discharging ears. He would wish to add an ear speculum and an electric light to the list of necessary instruments mentioned by Dr. Turner.

DR. S. F. MCDONALD said that it was impossible to differentiate between epilepsy and convulsions in children as clearly as in adults. Convulsive seizures were often related to a tendency to rickets in England and perhaps elsewhere. The Germans held that children were frequently intolerant of cows' milk; as the lime was excreted, the lack of calcium salts acted as a stimulant. Severe constipation produced by cows' milk was frequently noted in Brisbane.

DR. A. GRAHAM BUTLER, D.S.O., said that he grouped convulsions into three classes. In the first place, there were those associated with irritation from undigested food. The second class included convulsions in children with high fever, frequently due to toxic absorption from the intestines or to the onset of infective diseases. The third group comprised seizures caused by organic lesions of the cerebrum. He used opium more than chloral and carried small doses of Dover's powder ready for use. Turpentine was also of value for enemata.

DR. R. GRAHAM BROWN considered that the majority would agree that intoxication was the probable exciting cause of epilepsy. It was therefore essential in every case of epilepsy to hunt for a possible cause of the intoxication. He referred to patients who had ceased to have fits after a radical operation for the cure of a septic accessory sinus condition had been performed. The same occurred after the removal of septic tonsils or of an inflamed appendix. He considered that they were failing in their duty to their patient if they did not exclude all possible sources of auto-intoxication. He held that epilepsy would become less common as their investigations and treatment became more thorough.

Hay Fever.

DR. W. N. ROBERTSON, C.B.E., read a paper on "Hay Fever" (see page 207).

DR. GRAHAM BUTLER thought that hay fever and asthma should be treated together. It was an anaphylactic condition, though it was difficult to recognize the cause. Heredity seemed to play an important part. He had noted eosinophilia up to 70% in children.

DR. GRAHAM BROWN considered that hay fever and asthma were symptoms only, due to a variety of causes. The middle turbinate obstruction variety was the commonest. He thought that the incidence was higher in Brisbane than in England. He attributed this to the dry, dusty atmosphere of Brisbane. Females were more frequently affected than males. He agreed with Dr. Robertson that the condition became rarer after the age of twenty-five. He did not quite agree with the use of the cautery in all cases, but reserved it for patients in whom there was no obvious obstruction by the middle turbinate bone and in whom there was a slight increase in the blood pressure. He thought that this coincided with the views of Francis. While he held that there was an exciting cause for the onset of the symptoms in every case, he was equally insistent that there was also a predisposition to the affection in the patient. The middle turbinate could be enlarged without any symptoms of hay fever. The condition responsible for the symptoms was pressure, either outwards on the lateral wall of the nose, or inwards on the septum. The presence of certain bodies, such as dust, pollen and so forth, produced an irritation in this region, with a consequent swelling of the mucous membrane and a reflex sneeze or cough. He held that the essential point in the operation of submucous resection of the septum was not so much the straightening of the septum as the releasing of the pressure on the middle turbinate bone or bones. He therefore combined with this operation a partial middle turbinectomy, when there was suspicion of pressure in the directions indicated. He accepted Ballinger's hypothesis of the middle turbinate region being the vicious circle of the nose. He considered that the function of the middle turbinate was to protect the openings of the sinuses under its cover. The contention that its removal interfered with the sense of smell was incorrect. Haphazard cauterization of the nose by the general practitioner should be discountenanced. The mucous membrane of the nose was a highly specialized membrane, the scarring of which would lead to trouble. He had found that adrenalin, applied locally or subcutaneously, did not abolish the symptoms of asthma or hay fever in every case. He held that the removal of the inferior turbinate was rarely a justifiable operation. In conclusion, he complimented Dr. Robertson on his admirable paper.

In his reply, DR. ROBERTSON said that the removal of tonsils and adenoid vegetations often stopped convulsive seizures. Gentle expirations were good for asthma. The blood pressure in the majority of patients was not excessive. He had obtained good results in patients with low as well as those with high blood pressure. The catching of cold would be obviated by the removal of contact of the turbinates with the nasal wall. It did not follow that the treatment of asthma must be instituted in the early stages, for it was very difficult to treat children for this condition. Sinus trouble was a definite factor in asthma and hay fever and was often missed by the specialist who relied on trans-illumination and X-ray examination. It could frequently only be detected by the use of the trochar.

NOTICES.

THE COUNCIL OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION has arranged the following provisional programme of the Branch meetings. The Scientific Committee reserves to itself the right to modify the arrangements, but it is hoped that no changes will be necessary.

September 6, 1922.

CLINICAL MEETING at St. Vincent's Hospital, Melbourne, at 8.15 p.m..

October 4, 1922.

At the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne Hospital, at 8.15 p.m.

DR. H. DOUGLAS STEPHENS: "Clinical Symptoms of Enlarge- of the Spleen in Children."

DR. S. O. COWEN: "Familial Splenomegaly."

DR. K. HILLER will open the discussion.

November 1, 1922.

CLINICAL MEETING at the Children's Hospital, Carlton, at 8.15 p.m..

Medical Societies.

THE MEDICAL SCIENCES CLUB.

A MEETING of the Medical Sciences Club was held at the University of Adelaide on July 7, 1922.

Bilateral Anterior Polar Cataract.

DR. F. H. BEARE reported an instance of bilateral anterior polar cataract occurring in a child, aged four weeks. The condition had been noticed on the second day after birth. The mother had had the same condition from birth. She had no sisters and was unable to remember whether her mother had been similarly affected or not. Her brothers and her father, however, had displayed no abnormality. Dr. Beare said that Harman in "The Treasury of Human Inheritance" recorded only one family in which this condition had been displayed and that in this instance it had persisted through several generations. It was a very rare condition.

DR. R. H. PULLEINE agreed that the condiion was rare and said that he had only seen it once or twice in fifteen years.

DR. G. BULL pointed out that the ordinary form of cataract had long been regarded as hereditary in horses. He was unaware, however, of any basis for this supposition.

Pneumococcal Meningitis.

DR. F. H. BEARE, by permission of DR. GUNSON, also reported an instance of pneumococcal meningitis occurring in an infant six weeks old. The illness had begun eight days previouly. Four days after the onset convulsions had occurred and had continued at intervals until death. Lumbar puncture performed twice had yielded no fluid. The ventricle had been punctured on account of bulging of the fontanelle and a quantity of pus containing pneumococci had been obtained. *Post mortem* examination had shown the presence of a bilateral *otitis media*, the infection being purely pneumococcal. The whole of the surface of the brain had been found to be covered with pus, but no signs of infection had been discovered elsewhere. Dr. Beare said that two features in this case made it uncommon. In the first place there was the age of the patient and in the second the use of ventricular puncture, both for diagnostic and therapeutic purposes. There was no doubt that the child had been made much more comfortable as a result of the relief of pressure due to the puncture.

In answer to a question by Dr. Hayward, Dr. Beare could not say whether the *otitis media* had been a primary or a secondary condition. It had not appeared to be of long duration on account of the absence of perforation of the ear drums. There had been no pneumococcal infection elsewhere.

DR. PULLEINE reported having just seen a child, aged six weeks, with a double pneumococcal *otitis media*, but with as yet no brain involvement.

Disease in Australian Aborigines.

PROFESSOR J. B. CLELAND drew attention to the efforts that are being made by the Department of Pathology to secure all possible records of disease in aborigines. His attention had just been called to an interesting abnormality, discovered in the dissecting room and consisting

of a pouch or diverticulum near the pyloric end of the stomach, together with pronounced pyloric thickening which was not due to any malignant growth.

In a *post mortem* examination of an aboriginal who had died of tuberculosis, Professor Cleland had noticed an unusual reaction to the infection on the part of the tissues. The aborigines were supposed to have been isolated from other human beings for many thousands of years. Such a long isolation had not occurred with any other human race and, if it were true that the relative immunity of the white man to many infections was due to his constant exposure to these infections through many generations, then definite differences in the reaction of the tissues of the aboriginal to infection to those usual in white men might be expected. Hence records of all kinds of infectious diseases in the aboriginal should be carefully collected and preserved.

DR. W. RAY raised the question of the period of time necessary to acquire the immunity exhibited by the white man. He referred to a case that suggested the possibility of acquiring such an immunity in a single lifetime. This was the case of an aboriginal woman who had lived with white people ever since she had been removed from the aboriginal camp at the age of two years. In houses in which she had acted as domestic servant there had been, to the personal knowledge of Dr. Ray, epidemics of scarlet fever, measles, diphtheria and influenza. She had not contracted one of these complaints. Dr. Ray thought that this woman was fully as immune to these infections as any white person.

PROFESSOR CLELAND drew attention to the fact that when the first fleet reached Sydney the aborigines were already afflicted with small-pox. It did not appear possible that the infection had been introduced either by the first fleet or by the previous expedition of La Perouse. It was known that the Malays had first visited Australia in 1770 and Professor Cleland suggested that an epidemic introduced by them might have travelled across Australia and reached Sydney at about the time of the arrival of the first fleet. A subsequent epidemic among the aborigines of a very severe nature had been marked by the fact that it did not affect white men. In regard to other infections, bones recently found at Point MacLeay showed the presence of osteitis, which would ordinarily have been interpreted as due to syphilis. These bones, however, probably antedated the earliest white settlement. The origin of the lesions was still problematical, but it was possible that the aborigines were afflicted with this disease before the arrival of the white man. The infection must therefore have been introduced in another manner.

DR. RAY pointed out that by the end of the sixteenth century the northern coast had been tolerably well charted and that early explorers, Spanish and others, might have introduced syphilis in this region.

DR. PULLEINE said that in the sixteenth century Dutch navigators had been acquainted with the west coast of Australia. In these two ways, therefore, the white man might have been responsible for the introduction of this disease.

DR. H. C. NOTT stated that he possessed an aboriginal skull in which the frontal sinus was developed on one side only.

DR. PULLEINE said that he had seen the same abnormality in the skull of a white man.

The Administration of Digitalis.

DR. R. L. T. GRANT raised the question of the proper dose and mode of administering digitalis. He said that it had formerly been taught that dosage of more than 1.00 to 1.25 cubic centimetres (fifteen to twenty minims) of the tincture was dangerous and that it was usually stated that the effect took twenty-four hours to become manifest. Dr. Grant referred to the fact that Dr. Frazer, in England, had recently introduced intensive treatment with tincture of digitalis, giving as much as twelve cubic centimetres (three fluid drachms) in twenty-four hours. The first dose in such a case was six cubic centimetres, the second eight hours later being four and the third eight hours still later two cubic centimetres. This had resulted in slowing and strengthening the heart beat three hours after the first dose without any ill-effects. Dr. Frazer had stated that if there were danger, the patients averted it by vomiting the dose and that the method should only be used in hospitals where patients were continuously under observation.

DR. W. T. HAYWARD, C.M.G., remarked that he had always doubted the accuracy of the statement that digitalis took twenty-four hours to act. He had seen results much sooner if the digitalis were given in large doses.

DR. RAY had noticed the effect of digitalin in twelve hours, but the activity had varied greatly with the preparation used.

DR. G. BROWN said that he had seen seven doses of four to six cubic centimetres (one to one and a half fluid drachms) of the tincture used occasionally with good results obtained quickly.

Fireman's Cramp.

DR. R. L. T. GRANT drew attention to the condition seen on board ship and known as fireman's cramp. He stated that it was characterized by great abdominal pain, occurred in hot weather and was attributed by the sufferers to the drinking of large quantities of iced water.

DR. HAYWARD said that he had seen the condition in patients who had drunk large quantities of oatmeal water.

DR. PULLEINE reported having seen a case which occurred in a miner in a gold mining town in Queensland. The patient had drunk large quantities of oatmeal water in very hot weather and after exertion. The onset of pain had been sudden and the patient had taken a dose of rum, which had resulted in acute dilatation of the stomach and subsequent death.

DR. GRANT said that the condition as seen by him had not been accompanied by dilatation of the stomach, but had consisted of acute colic without diarrhœa.

Muscle Fibres of Nasonia Brevicornis.

MR. TIEGS showed sections of muscle fibres obtained from the head muscle of a parasitic wasp (*Nasonia brevicornis*). The transverse striation in the fibrils and Krause's membrane could be clearly seen. The transverse striations consisted of dumb-bell shaped thickenings, the thin part of the dumb-bell being known as "Henson's line." Sometimes the striations were seen to run in a double spiral round the fibrils.

Toxic Conditions.

DR. RAY drew attention to the administration *per rectum* of constant saline drips saturated with glucose, a therapeutic procedure which he regarded as of great value in toxic conditions. For some time past it had been his custom to treat all patients in hospital suffering from pneumonia in this fashion. Dr. Ray said that this addition to the fluid contents of the body reduced the toxicity and that through the use of this method he had seen recovery occur in the case of patients whom he would otherwise have expected to die. He thought that this method of treatment should be more widely known. No serious complication was met with, provided that the rate of drip was carefully adjusted to be equal to the rate of absorption. Its use was contraindicated in typhoid fever. It might be given without any disadvantage if slight albuminuria were present, but it had not been administered to patients suffering from arterio-sclerosis.

The Effect of Tropical Climate.

PROFESSOR T. BRAILSFORD ROBERTSON drew attention to articles by Dr. E. S. Sundström appearing in a recent issue of the *American Journal of Physiology*. These articles embodied the results of very extensive investigations on the effect, both immediate and ultimate, of an artificially induced tropical climate on animals bred therein up to the fourth generation. This method of investigating the effects of tropical climate was especially important, because it enabled distinctions to be made between those effects which were truly climatic in origin and those which were secondary to parasitic infection.

Correspondence.

INCOME TAX.

SIR: In your issue of August 12, in answer to a question on the above subject by "Professional," the more generous deduction of 12½% is given than that actually allowed by the Federal authorities, which is the rate (10%) advised by experts in a recent issue of your journal and that only on yearly depreciated value and not on original cost. Thus a £500 car would still at the end of twenty years have by the Department's rate a depreciated value of £60!

The State Tax in New South Wales allows 20% depreciation as being "a fair deduction," while a recent explanation by the Federal Department reads: "No allowance can be made on account of loss on sale of an old car as the Departmental rates of depreciation, based on the diminishing value of an asset, are only intended to cover depreciation due to wear and tear and not fully covered by allowance of repairs and maintenance. Upon the realization of an asset the difference between the amount of realized and the written-down value, based on Departmental rates, is regarded as a capital loss, being actual depreciation due to obsolescence, etc., and not wear and tear."

As this is a serious matter to the professional owners of high-priced cars, which are an absolute essential in practice (especially country ones) it might help to a more generous and equitable deduction being allowed if the Federal Committee of the Association would move in the matter and even obtain the allowance granted by the New South Wales State Department.

Yours, etc.,

"COUNTRY PRACTITIONER."

August 11, 1922.

DRIED MILK.

SIR: I agree with Dr. Graham Butler that in the present conditions of commercial milk production and distribution, milk powder is preferable for infant feeding, particularly in the hot weather. We have been reluctantly compelled to accept the lesser of two evils. The manufacturers of "Glaxo" and "Lactogen" have at last adopted my suggestions, made long ago, to alter the directions on the tin in conformity with the requirements of a semi-tropical climate and they enclose an accurate metal measure with the package to obviate the necessity of using the unreliable household spoon. Previously thousands of artificially-fed infants suffered from severe digestive disorders owing to gross overfeeding with a perfectly good food. Granted that dried milks in use here are produced under ideal conditions and the instructions for preparation are as correct as may be, everyone must surely admit they are only a compromise, not perfection. Argue as we may, we know at heart that pure fresh cows' milk in the truest sense is superior in every way to the tinned stuff. Dried milks are perhaps acceptable to the dumb, uncomplaining infant, but are unpalatable and often actually nauseous to children and adults. One can hardly imagine a normal youngster, home from school, drinking a large glass of milk powder concoction with relish. *De gustibus non disputandum* and taste and appetite are of considerable importance as affecting absorption and metabolism. The habit of milk drinking should be acquired and developed by every member of the community, when there is no idiosyncrasy. It has been well said that "Milk is the most satisfactory single article of food which is suitable for consumption by man." Milk and leafy vegetables are the two paramount "protective foods," compensating the deficiencies of and supplementing other foods in actual elements, quite apart from enzymes and vitamins.

Dr. Graham Butler states that definite cases of deficiency diseases are rare nowadays, but he entirely overlooks the fact that what we don't know about them would fill a library. Advanced degrees are certainly unusual in this land of sun and open air, but what about the "sub-clinical" stages? McCollum, "The Newer Knowledge of Nutrition," 1922, says on page 206: "Many grades of injury less severe than the clinically recognizable deficiency diseases. . . . It is becoming more and more evident that even slight departure from the optimal in the composition of the food may lead to states of nutritional instability which become contributing factors to physical breakdown when hygienic factors are unfavourable or when infectious processes are operating." And, again, on page 217, that: "Borderline malnutrition, not causing alarm, is of greater aggregate importance than deficiency diseases. . . . We are taking too little of the protective foods—milk and the leafy vegetables—and a movement toward stimulating the consumption of these classes of foods would go far toward effecting improvement in our national health standards." He also points out that the experimental studies of dried milks in reference to vitamin values are so inconclusive and divergent in results that no reliance can be placed on them. We do, however, know what fresh milk accomplishes and we know it is unique. Instead of a pessimistic, supine attitude as a profession we must breed a healthy discontent and demand a milk supply that is pure, without purification. We must insist on perfection till we get it. I hope to report fully before long a method of milking that makes the exclusion of organisms an easy matter, provided simple common sense is employed. If this can be done without increased cost, it should be made illegal to sell contaminated or impure milk, using regular bacterial counts as an indicator. The one essential is scruplous cleanliness in every stage of handling; it come immeasurably before food value and we must agree with Charlie Reade: "It's never too late to mend."

Yours, etc.,

SELWYN HARRISON.

Sydney, August 15, 1922.

Obituary.

JOSEPH HINES WOLFENDEN.

AFTER almost a half century of medical practice in Victoria, Dr. J. H. Wolfenden passed away at Malvern on July 5, 1922.

Joseph Hines Wolfenden was born in County Tipperary, Ireland, and was the son of the Reverend Henry Bingham Wolfenden, of Kilfinaghty Glebe, Bunratty, Ireland.

His medical course covered the period 1869 to 1873 and was taken at King and Queen's College of Physicians and the Royal College of Surgeons, Dublin. A prizeman in *materia medica*, he also attained distinction and was awarded special certificates in anatomy, eye and ear work and obstetrics and gynæcology.

After a term of four months as house surgeon at the Adelaide Hospital, Dublin, Joseph Hines Wolfenden joined his brother as an assistant in a country practice in England. Towards the end of 1874 he came to Australia and found his way to Dunnolly, to act as *locum tenens* for another brother who was in practice in that centre.

Joseph Hines Wolfenden elected to settle in Dunnolly, was appointed resident surgeon to the Dunnolly District Hospital and practised in the township and outlying district for the long and uninterrupted spell of forty years. He retired early in 1915, since when he has lived at Malvern and restricted his practice in order that he might have comparative rest in his later years.

He received a commission in the Victorian Military Forces as Surgeon-Captain in 1890, but for some years preceding his death was on the retired list.

Joseph Wolfenden was attracted by Freemasonry and was a member of the craft for many years. His unfailing resort in his spare time was his garden, where he devoted many hours to labour which was altogether a delight.

A widow and three daughters survive him.

Naval and Military.

APPOINTMENTS.

THE following appointments, promotions, etc., have been notified in the *Commonwealth of Australia Gazette*, Nos. 53, 58, 60 and 61, of June 22, July 13 and 27 and August 3, 1922:

Australian Military Forces.

FIRST MILITARY DISTRICT.

Reserve of Officers.

HONORARY MAJOR H. J. STEWART is transferred from the Reserve of Officers, Second Military District, 1st June, 1922.

SECOND MILITARY DISTRICT.

Australian Army Medical Corps.

COLONEL K. SMITH, C.M.G., is transferred to the Reserve of Officers, 1st July, 1922.

CAPTAIN J. C. DOUGLAS is transferred to the Reserve of Officers, 1st June, 1922.

Australian Army Medical Corps Reserve.

To be Honorary Captain—ARTHUR STANLEY HUGHES, 1st June, 1922.

Reserve of Officers.

HONORARY MAJOR H. J. STEWART is transferred to the Reserve of Officers, 1st Military District, 1st June, 1922.

THE notification respecting the grant of the substantive rank of Major to HONORARY CAPTAIN A. M. LANGAN, Australian Army Medical Corps Reserve, which appeared in Executive Minute No. 175/1921, promulgated in *Commonwealth of Australia Gazette*, No. 43, dated 12th May, 1921, is cancelled.

MAJOR A. P. DRUMMOND is transferred from the Reserve of Officers, Third Military District, 1st July, 1922.

CAPTAIN J. H. R. MCCUTCHEON is transferred from the Reserve of Officers, Sixth Military District, 1st June, 1922.

To be Major—CAPTAIN C. COSGROVE, M.C., 1st June, 1922.

To be Captains—HENRY CHARLES BARRY, M.C., 1st October, 1920 (this cancels the notifications respecting this officer which appeared in Executive Minutes Nos. 175/1921 and 206/1921, promulgated in *Commonwealth of Australia Gazette*, Nos. 43, dated 12th May, 1921, and 53, dated 16th June, 1921, respectively); HONORARY CAPTAIN RUPERT DUFFY HEGGATON, 1st January, 1921 (this cancels the notification respecting this officer which appeared in Executive Minute No. 64/1922, promulgated in *Commonwealth of Australia Gazette*, No. 17, dated 2nd March, 1922.)

THE notification respecting the grant of the honorary rank of Captain to C. COSGROVE, M.C., which appeared in Executive Minute No. 518/1919, promulgated in *Commonwealth of Australia Gazette*, No. 92, dated 24th July, 1919, is cancelled.

THIRD MILITARY DISTRICT.

Australian Army Medical Corps.

THE resignation of LIEUTENANT E. H. DERRICK of his provisional appointment is accepted, 19th May, 1922.

Reserve of Officers.

To be Captains—HONORARY CAPTAIN JOHN RICHARDS HARRIS, Australian Army Medical Corps Reserve, 1st October, 1920 (this amends the notification respecting this officer which appeared in Executive Minute No. 176/1921, promulgated in *Commonwealth of Australia Gazette*, No. 43, dated 12th May, 1921); ANDREW JAMES DWYER, D.S.O., 1st January, 1922; HONORARY CAPTAIN S. M. WARE, 1st January, 1921 (this cancels the notification respecting this officer which appeared in Executive Minute No. 437/1921, promulgated in *Commonwealth of Australia Gazette*, No. 88, dated 10th November, 1921); HONORARY CAPTAIN F. F. A. L. LEMON, Australian Army Medical Corps Reserve, 1st January, 1921 (this cancels the notification respecting this officer which appeared in Executive Minute No. 85/1922, promulgated in *Commonwealth of Australia Gazette*, No. 26, dated 23rd March, 1922).

CAPTAIN F. G. HEYER is transferred from the Reserve of Officers, Sixth Military District, 1st June, 1922.

MAJOR A. P. DRUMMOND is transferred to the Reserve of Officers, Second Military District, 1st July, 1922.

HONORARY CAPTAIN R. H. CRISP is transferred to the Reserve of Officers, Fifth Military District, 1st July, 1922.

HONORARY CAPTAIN A. SANDISON to be transferred to the Reserve of Officers, 4th Military District, 1st July, 1922.

Australian Army Medical Corps Reserve.

THE notification respecting the appointment of SAMUEL MIDDLETON WARE as Honorary Captain, which appeared in Executive Minute No. 275/1916, promulgated in *Commonwealth of Australia Gazette*, No. 40, dated 30th March, 1916, is cancelled.

FOURTH MILITARY DISTRICT.

Reserve of Officers.

To be Honorary Captain—MALCOLM LESLIE SCOTT, 22nd May, 1916. (This cancels the notification respecting this officer which appeared in Executive Minute No. 347/1919, promulgated in *Commonwealth of Australia Gazette*, No. 57, dated 15th May, 1919.)

To be Captains—HONORARY CAPTAIN M. L. SCOTT, 1st October, 1920 (this cancels the notification respecting this officer which appeared in Executive Minute No. 599/1920, promulgated in *Commonwealth of Australia Gazette*, No. 113, dated 23rd December, 1920); HONORARY CAPTAIN JOHN NEWTON WEBB, Australian Army Medical Corps Reserve, 1st January, 1920 (this amends the notification respecting this officer which appeared in Executive Minute No. 105/1921, promulgated in *Commonwealth of Australia Gazette*, No. 24, dated 17th March, 1921); HONORARY CAPTAIN GEOFFREY HOWARD BARHAM BLACK, Australian Army Medical Corps Reserve, 1st July, 1920 (this amends the notification respecting this officer which appeared in Executive Minute No. 23/1921, promulgated in *Commonwealth of Australia Gazette*, No. 6, dated 13th January, 1921).

THE notification respecting the grant of the substantive rank of Captain to G. H. B. BLACK, which appeared in Executive Minute No. 191/1920, promulgated in *Commonwealth of Australia Gazette*, No. 36, dated 22nd April, 1920, is cancelled.

HONORARY CAPTAIN A. SANDISON to be transferred from the Reserve of Officers, Third Military District, 1st July, 1922.

THE notification respecting the grant of the substantive rank of Captain to F. M. WILCOX, which appeared in Executive Minute No. 191/1920, promulgated in *Commonwealth of Australia Gazette*, No. 36, dated 22nd April, 1920, is cancelled.

Australian Army Medical Corps Reserve.

THE name of GEOFFREY HOWARD BARHAM BLACK is as now stated and not as shown in Executive Minute No. 916/1916, promulgated in *Commonwealth of Australia Gazette*, No. 154, dated 26th October, 1916.

FIFTH MILITARY DISTRICT.

Australian Army Medical Corps.

LIEUTENANT (provisionally) R. H. MORGAN is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 12th July, 1922.

MAJOR C. H. SHEARMAN and CAPTAIN A. BADOCK to be transferred to the Reserve of Officers, 1st July, 1922.

SIXTH MILITARY DISTRICT.

Reserve of Officers.

To be Captain—DOUGLAS BINGHAM WHEELER, M.B.E., 1st January, 1920. (This cancels the notification respecting this officer which appeared in Executive Minute No. 134/1920, promulgated in *Commonwealth of Australia Gazette*, No. 29, dated 18th March, 1920.)

CAPTAIN F. G. HEYER is transferred to the Reserve of Officers, Third Military District, 1st June, 1922.

CAPTAIN J. H. R. MCCUTCHEON is transferred to the Reserve of Officers, Second Military District, 1st June, 1922.

Australian Army Medical Corps.

CAPTAIN L. GOLLAN to be transferred to the Reserve of Officers, 1st July, 1922.

CAPTAIN (provisionally) E. A. ELLIOTT to be transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st July, 1922.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of the *Medical Act, 1912 and 1915*, as duly qualified medical practitioners:

DAVISON, SAMUEL HOWARD, M.B., Ch.B., 1912; M.D., 1916, Q.U., Belfast, 16, Enmore Road, Newtown.

HARTNETT, LEONARD JOHN THOMAS, M.B., Bac. Surg., 1918 (Univ. Melbourne), Wodonga.

HEBBERT, CHARLES ALFRED, M.R.C.S. (Eng.), 1880; M.R.C.P. (Lond.), 1884, 70, Gerard Street, Cremorne.

MASON, JOHN WHARTON, M.B., Ch.B., 1921 (Univ. Edin.), Tumut.

Additional Registration.

LOWE, GORDON BRADLEY, F.R.C.S. (Edin.), 1921.

Books Received.

A SYSTEM OF CLINICAL MEDICINE, DEALING WITH THE DIAGNOSIS, PROGNOSIS AND TREATMENT OF DISEASE, by Thomas Dixon Savill, M.D. (Lond.); Sixth Edition; 1922. London: Edward Arnold & Company; Demy 8vo., pp. 923, and index, with four plates in colour and 172 illustrations in the text. Price: 28s. net.

APPLIED CHEMISTRY: AN ELEMENTARY TEXT-BOOK FOR SECONDARY SCHOOLS, by Fredus N. Peters, Ph.D.; 1922. St. Louis: C. V. Mosby Company; Post 8vo., pp. 461, with 72 illustrations. Price: $3.50.

CHLOROFORM ANÆSTHESIA, by A. Goodman Levy, M.D., M.R.C.P., with a foreword by Arthur R. Cushny, M.D., LL.D., F.R.S.; 1922. London: John Bale, Sons & Danielsson, Limited; Demy 8vo., pp. 159, with nine figures. Price: 7s. 6d. net.

ENDOCRINE THERAPEUTICS: PRACTICAL SUGGESTIONS, by Thomas Bodley Scott; 1922. London: H. K. Lewis & Company, Limited; Crown 4to., pp. 118. Price: 5s. net.

OTO-RHINO-LARYNGOLOGY FOR THE STUDENT AND PRACTITIONER, by Dr. Georges Laurens; Authorized English Translation of the Fourth Revised French Edition, by H. Clayton Fox, F.R.C.S. (Irel.), with a foreword contributed by Sir J. Dundas-Grant, M.A., M.D., F.R.C.S.; The Second English Edition; 1922. Bristol: John Wright & Sons, Limited; Demy 8vo., pp. 333, with index, and 589 illustrations in the text. Price: 17s. 6d. net.

PHYSICAL EXAMINATION OF THE CHEST, WITH SPECIAL REFERENCE TO PULMONARY TUBERCULOSIS, INCLUDING A CHAPTER ON TUBERCULOSIS OF THE LARYNX, by James Crocket, M.D., D.P.H., M.R.C.P.E.; 1922. London: H. K. Lewis & Company, Limited; Crown 8vo., pp. 254, with 56 illustrations, including 14 plates. Price: 9s. net.

SYPHILIS, PALUDISME AMIBIASE: LE TRAITEMENT D'ATTAQUE ET LES TRAITEMENTS SECONDAIRES, par le Dr. Paul Ravaut; Préface du Professeur Fernand Widal; Deuxième édition refondue; 1922. Paris: Masson et Cie; Post 8vo., paper cover, pp. 214. Price: Frs. 9 net.

Medical Appointments.

DR. REGINALD WEBSTER (B.M.A.) has been appointed a Member of the Queen's Memorial Infectious Diseases Hospital Board, Victoria.

* * * *

DR. H. R. J. HARRIS (B.M.A.) has been appointed Government Medical Officer at Deepwater, New South Wales.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

ADELAIDE HOSPITAL: Vacancy for Laboratory Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Societies Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Hampden District Hospital, Kuridala, North Queensland Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

AUG. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
AUG. 24.—Brisbane Hospital for Sick Children: Clinical Meeting.
AUG. 25.—New South Wales Branch, B.M.A.: Branch.
AUG. 25.—Queensland Branch, B.M.A.: Council.
AUG. 30.—Victorian Branch, B.M.A.: Council.
AUG. 31.—South Australian Branch, B.M.A.: Branch.
SEPT. 1.—Queensland Branch, B.M.A.: Branch.
SEPT. 6.—Victorian Branch, B.M.A.: Branch.
SEPT. 7.—New South Wales Branch, B.M.A.: Nomination of Candidates for Federal Committee.
SEPT. 8.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 8.—Queensland Branch, B.M.A.: Council.
SEPT. 8.—South Australian Branch, B.M.A.: Council.
SEPT. 9.—Eastern District Medical Association, New South Wales.
SEPT. 12.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 13.—Western Australian Branch, B.M.A.: Council.
SEPT. 13.—Melbourne Pædiatric Society.
SEPT. 14.—Victorian Branch, B.M.A.: Council Meeting.

Editorial Notices.

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All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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